

**EFFECT OF AN AGGRESSIVE VERSUS CONSERVATIVE, MULTI-MODAL  
REHABILITATION PROGRAMME ON CHRONIC LOWER BACK PAIN**

by

**J.H. BILLSON**

submitted in partial fulfillment of the requirements for the degree

**DOCTOR PHILOSOPHIAE**

in the

**FACULTY OF HUMANITIES**

**(Department of Biokinetics, Sport and Leisure Science)**

**University of Pretoria**

Promoter: Prof. P.E. Kruger

October 2009

## DEDICATION

I dedicate this thesis to my late grandfather, John Henry Billson. His passion for knowledge and his driving to understand has been an inspiration for me through the years. The opportunity to complete a thesis was never open to him, but it was his desire for his grandchildren to complete that which he was never given the opportunity to do. This has become the desire of my heart. My only regret is that he is not with us to see me achieve a goal that we both share.

According to the old saying, it is better to travel hopefully than to arrive. Our quest for discovery fuels our creativity in all fields, not just science. If we reached the end of the line, the human spirit would shrivel and die. But I don't think we will ever stand still; we shall increase in complexity, if not in depth, and shall always be the center of an expanding horizon of possibilities.



*Prof. Stephen Hawking in: The Universe in a Nutshell  
Lucasian Professor of Mathematics at the University of Cambridge and  
regarded as one of the most brilliant theoretical physicists since Einstein.*

## ACKNOWLEDGEMENTS

I wish to make the following acknowledgements:

**To the Almighty Father:** For granting me strength and blessing me with more than any person deserves. I am but his humble servant. All glory to God.

**Prof. P.E. Krüger:** My presenter, for showing faith in me as a person and a student, and for guiding my work with great care and wisdom.

**Dr. B. van Vuuren:** For initially guiding my research and all the effort that went into starting the project.

**Malize Alexander:** For her assistance with the statistical analysis of the data.

**The University of Pretoria:** For their financial assistance and opportunities.

**Jansie Louw:** For all of her unbelievable patience, kindness and willingness to help in gathering all of the many research articles that were used in this study.

**My wife Frieda:** Her love and patience with me and my work have been support that I could not do without. She is the light of my life and a shining beacon of hope.

**My parents:** For all of their patience, love and support throughout the years, and who have made it possible for me to complete this study. Without them I would be nothing.

## SYNOPSIS

<b>Title</b>	<b>Effect of an aggressive versus conservative, multi-modal rehabilitation programme on chronic lower back pain</b>
<b>Candidate</b>	<b>John Henry Billson</b>
<b>Presenter</b>	<b>Prof. P.E. Krüger</b>
<b>Department</b>	<b>Biokinetics, Sport and Leisure Science</b>
<b>Degree</b>	<b>Doctor Philosophiae</b>

Low back pain has become one of the most influential musculoskeletal diseases of modern society. It is one of most expensive diseases in terms of medical costs and increased worker absenteeism, which can lead to permanent disability and places strain on the economy as a whole. Pain has been recognised as a disease in itself, which has certain consequences when it becomes chronic. Many kinds of treatment options exist with varying degrees of success. The question is thus which treatment option is the most favourable and cost-effective.

Conservative treatment is the most recommended form of treatment when no serious underlying diseases are present. Exercise has been shown to be very effective in the treatment of chronic low back pain but there are still questions regarding the use of exercise therapy.

The predetermined goal of the study was to ascertain whether an aggressive-progressive exercise programme, and specifically what kind of exercises, would be more effective in the treatment of chronic low back pain. This was achieved through a number of steps, which included an extensive literature review, the identification of an appropriate test battery with related minimum physical requirements and cut scores, subject recruitment and screening of subjects, the implementation of the intervention and the subsequent re-testing of the subjects.

Once the data was completed, the next step was to make use of two case

studies to assist in illustrating the effectiveness of individual patients compared to the sample as a whole. These case studies were of patients who completed the entire programme but one took longer to complete the programme. This assists in illustrating the value of maintaining exercise protocol.

The results from the present study are extremely positive. The two case studies provided a glimpse of the potential value that could be added through the implementation of more aggressive-progressive exercise interventions in the treatment of chronic low back pain. The final product will greatly assist exercise therapists concerned with the treatment of chronic low back pain along with cognitive-behavioural techniques. Hopefully this study will provide insight into managing chronic low back pain in South Africa from an exercise standpoint. Secondly the study will provide practical techniques to implement in an era in which economic difficulties are rife.

### **Keywords**

Aggressive-progressive exercises

Full working capacity adults

Cognitive-behavioural techniques

Multidisciplinary/interdisciplinary

Neuropathic pain

Fear avoidance behaviour

Disability

Work absenteeism

## SAMEVATTING

<b>Titel</b>	<b>Invloed van 'n aggressiewe versus 'n konserwatiewe, multimodale rehabilitasieprogram op chroniese laerugpyn</b>
Kandidaat	John Henry Billson
Promotor	Prof. P.E. Krüger
Departement	Biokinetika, Sport- en Vryetydswetenskappe
Graad	Doctor Philosophiae

Laerugpyn het een van die invloedrykste muskuloskeletale siektes van die moderne samelewing geword. Dit is een van die duurste siektes in terme van mediese koste en verhoogde siekverlof deur werkers, wat kan lei tot permanente ongeskiktheid en 'n verhoogde las plaas op die ekonomie as 'n geheel. Pyn word erken as 'n siekte op sy eie wat sekere gevolge het wanneer dit chronies begin raak. Verskeie soorte behandelingsopsies is beskikbaar met variërende grade van sukses. Die vraag is dus watter behandelingsopsie is die bruikbaarste en koste-doeltreffendste.

Konserwatiewe behandeling is die mees aanbevole metode van behandeling wanneer daar geen ernstige onderliggende siektetoestande teenwoordig is nie. Dit is reeds bewys dat oefening baie doeltreffend is in die behandeling van chroniese laerugpyn. Daar bestaan egter steeds vrae rondom die gebruik van oefening as terapie.

Die vooropgestelde doelwit van die studie was om te bepaal of 'n aggressiewe-progressiewe inoefeningsprogram doeltreffend sal wees in die behandeling van chroniese laerugpyn, en meer spesifiek watter tipe oefening die doeltreffendste sal wees. Die navorsing het bestaan uit 'n paar stappe wat ingesluit het 'n intensiewe literatuursoektog, die identifisering van 'n gepaste toetsbattery met verwante minimum fisieke vereistes en afsnytellings, die verkryging en evaluering van proefpersone, die implementering van die intervensieprogram en die daaropvolgende hertoetsing van die proefpersone.

Nadat die invordering van die data en die gepaardgaande analise van die data voltooi is, was die volgende stap om gebruik te maak van twee gevallestudies ten einde die doeltreffendheid van die intervensieprogram vir individuele proefpersone te illustreer deur dit te vergelyk met die groep as 'n geheel. Die twee gevallestudies was van proefpersone wat die intervensieprogram volledig voltooi het, alhoewel die een proefpersoon langer geneem het om die intervensieprogram te voltooi. Dit help om die navolgingswaarde van 'n inoefeningsprotokol te illustreer.

Die resultate van die huidige studie is uiters positief. Die twee gevallestudies gee 'n mate van insig wat betref die potensiële waarde wat verkry kan word deur die implementering van 'n meer aggressiewe-progressiewe inoefeningsintervensie vir die behandeling van chroniese lae rugpyn. Die finale produk sal die nodige ondersteuning aan oefeningsterapeute bied wat onseker is oor die behandeling van chroniese laerugpyn deur middel van aggressiewe-progressiewe inoefeningsintervensies en kognitiewe gedragstegnieke. Hierdie studie sal dus die begrip en insig van die behandeling van chroniese laerugpyn in Suid-Afrika verhoog vanuit 'n oefeningsuitgangspunt. Tweedens sal die studie die gebruik van praktiese oefentegnieke aanmoedig in 'n era waarin ekonomiese tye moeilik is.

### **Sleutelsterme**

Aggressiewe-progressiewe oefening

Volwerkendekapasiteit-volwassenes

Kognitiewe gedragsmetode

Multidissiplinêre/interdissiplinêre

Neuropatiese pyn

Vreesvermydingsgedrag

Ongeskiktheid

Werkafwesigheid

## TABLE OF CONTENTS

	<b>Page #</b>
DEDICATION	i
ACKNOWLEDGEMENTS	ii
SYNOPSIS	iii
SAMEVATTING	v
TABLE OF CONTENTS	vii
LIST OF TABLES	xiv
LIST OF FIGURES	xv
LIST OF EXERCISE PHOTOS	xvii
LIST OF ABBREVIATIONS	xix

---

### CHAPTER 1: THE PROBLEM

---

1.1	Introduction	1
1.2	Research Questions	7
1.3	Research Hypothesis	7
1.4	Goals of the Study	8
1.5	Objectives of the Study	8
1.6	Research Design	9
1.7	Research Procedure and Strategy	10
1.7.1	Inclusion Criteria	11
1.7.2	Exclusion Criteria	11
1.7.3	Study Sample	11
1.7.4	Intervention	12
1.7.5	Back School	13
1.8	Definition of Key Concepts	13



---

## CHAPTER 2: LITERATURE SURVEY

---

2.1	Pain and its Physiology	16
2.1.1	The Pain System	18
2.1.2	Types of Pain	21
2.1.2.1	Nociceptive Pain	21
2.1.2.1.1	Peripheral Mechanisms of Pathophysiological Nociceptive Pain	23
2.1.2.2	Neuropathic Pain	24
2.1.2.2.1	Peripheral Mechanisms of Neuropathic Pain	25
2.1.2.3	Dysfunctional Pain	26
2.1.2.4	Mixed Pain	27
2.1.3	Chronic Pain and its Effects	27
2.1.3.1	Psychological/Psychosocial Consequence of Chronic Low Back Pain	30
2.1.3.2	Physical Changes and Deconditioning	35
2.1.3.3	The Concept of Central Sensitisation	40
2.1.3.3.1	The Role of Acute Pain in Central Sensitisation	40
2.1.3.3.2	The Physiology of Central Sensitisation	41
2.2	The Problem of Low Back Pain	43
2.2.1	Lifetime Occurrence of Low Back Pain	45
2.2.2	Impact of Low Back Pain	46
2.3	Low Back Anatomy	48
2.3.1	The Bone Structure	48
2.3.2	The Intervertebral Disc	51
2.3.2.1	The Nucleus Pulposus	52
2.3.2.2	Annulus Fibrosis	53
2.3.2.3	The Endplate	54
2.3.2.4	Properties and Function of the Intervertebral Discs	54
2.3.3	Movements of the Vertebral Column	56

2.3.4	The Model of Spinal Stability and Instability	56
2.3.5	The Passive Stabilising Structures of the Spine: Bone, Ligaments and Fascia	58
2.3.6	Dynamic Spine Stability: The Muscles Supporting the Spine	59
2.3.6.1	Muscles Involved in Spinal Stability	64
2.3.6.1.1	Multifidus	68
2.3.6.1.2	Transversus Abdominis	73
2.3.6.1.3	Internal and External Obliques	75
2.3.6.1.4	Quadratus Lumborum	78
2.3.6.1.5	Erector Spinae (Extensor Group)	79
2.3.6.1.6	Gluteus Maximus, Gluteus Medius, and Gluteus Minimus	80
2.3.6.1.7	Rotatores and Intertransversarii	81
2.3.6.1.8	Rectus Abdominis	81
2.3.6.1.9	Latissimus Dorsi	82
2.3.6.2	Neuromuscular Stabilisation and Postural Control	83
2.3.6.3	The Role and Application of Stabilisation	86
2.4	Recommended Treatment Modalities for Low Back Pain	89
2.4.1	Acute Low Back Pain and its Necessity for Exercise Treatment	90
2.4.2	Recommended Treatment for Chronic Low Back Pain	92
2.4.3	The Role of Exercise as a Treatment Modality	97
2.4.4	The Use of Exercise Intervention in Chronic Low Back Pain	100
2.4.5	Conservative vs. Aggressive Exercise Treatments	105
2.4.5.1	Does Aggressive Exercise Rehabilitation Play a Role in Managing Chronic Low Back Pain?	106
2.5	Ergonomics: The Key to Protecting the Spine	106
2.5.1	The Role of Ergonomic Modification and Risk Factor Prevention	107

2.5.2	Specific Task Modification: Occupational Risk Factor Management	109
2.5.3	The Back School Concept: The Role of Research and its Application	110
2.6	Research Problem	114

---

## CHAPTER 3: METHODOLOGY

---

3.1	Introduction	116
3.2	Participants	116
3.2.1	History of the Subjects	117
3.2.1.1	The Use of Low Numbers in the Present Study	118
3.3	Methods and Materials	121
3.3.1	Medical Screening	121
3.3.2	Study Design	121
3.3.3	Questionnaires	122
3.3.3.1	Pain and Disability	122
3.3.3.1.1	The Visual Analogue Scale (VAS) for Pain Measurement	122
3.3.3.1.2	Oswestry Disability Index (ODI)	123
3.3.3.1.3	Functional Rating Index (FRI)	123
3.3.3.2	Fear Avoidance	124
3.3.3.2.1	Fear Avoidance Beliefs Questionnaire (FABQ)	124
3.3.3.3	Kinesiophobia	125
3.3.3.3.1	The Tampa Scale of Kinesiophobia	125
3.3.3.4	Exercise Intensity	125
3.3.3.4.1	The Borg Rate of Perceived Exertion (RPE) Scale	125
3.3.4	Physical Testing	127
3.3.4.1	Neurodynamic Testing	127
3.3.4.1.1	Straight Leg Raise Test	127

3.3.4.1.2	The Slump Test	131
3.3.4.2	Muscle Endurance Testing	132
3.3.4.2.1	The Sorenson Back Extension Test (The Ito Test Version)	132
3.3.4.2.2	Side Bridging Endurance Test	136
3.3.4.2.3	Flexor Endurance Test	137
3.3.5	The Exercise Programmes	138
3.3.5.1	Control Group (Conservative Exercise Programme)	138
3.3.5.2	Experimental Group (Progressive-Aggressive Programme)	141
3.3.5.2.1	Programme 1	141
3.3.5.2.2	Programme 2	144
3.3.5.2.3	Programme 3	148
3.3.6	The Back School	151

---

## CHAPTER 4: RESULTS AND DISCUSSION

---

4.1	Background and Objectives	154
4.2	Research Design	154
4.3	Methodology	154
4.4	Statistical Analysis	155
4.4.1	Descriptive Statistics	155
4.4.2	Inferential Statistics	156
4.4.2.1	Mann-Whitney Test	156
4.4.2.2	Wilcoxon Signed-rank Test	156
4.5	Results	156
4.5.1.	Descriptive Statistics for the Two Groups on all Measurements	157
4.5.2	Frequency, Intensity and Duration	171

4.5.3	Results of Differences Between Experimental and Control Groups on Pre-test Measurements	186
4.5.4	Results of Differences Between Experimental and Control Groups on Post-test Measurements	189
4.5.5	Results of the Analysis to Test Whether Statistically Significant Differences Existed Between the Pre-test and Post-test Measurements Within the Experimental Group	191
4.5.6	Results of the Analysis to Test Whether Statistically Significant Differences Existed Between the Pre-test and Post- test Measurements Within the Control Group	193
4.6	Case Studies	196
4.6.1	Patient A	196
4.6.1.1	Pre-test Results	196
4.6.1.2	Post-test Results	197
4.6.2	Patient B	198
4.6.2.1	Pre-test Results	198
4.6.2.2	Post-test Results	199

---

## **CHAPTER 5: SUMMARY, CONCLUSION AND RECOMMENDATIONS**

---

5.1	Introduction	201
5.2	Summary of Results	201
5.3	Conclusion	204
5.4	Recommendations	207
5.5	Future Research	209



<b>BIBLIOGRAPHY</b>	213
<b>ANNEXURE A</b>	306
<b>ANNEXURE B</b>	317
<b>ANNEXURE C</b>	329
<b>ANNEXURE D</b>	338

## LIST OF TABLES

<b>Table</b>		<b>Page #</b>
3.1	Borg RPE Scale	126
4.1	Descriptive Statistics per Group on Pre-test Measurements	157
4.2	Descriptive Statistics per Group on Pre-test and Post-test Measurements	159
4.3	Descriptive Statistics per Group on Pre-test and Post-test Measurements (continued)	161
4.4	Descriptive Statistics per Group on Pre-test and Post-test Measurements (continued)	164
4.5	Descriptive Statistics for the Experimental Group on Questionnaire Measurements	169
4.6	Descriptive Statistics for the Experimental Group on Questionnaire (continued)	170
4.7	Descriptive Statistics for the Experimental and Control Groups (continued)	174
4.8	Descriptive Statistics for the Experimental and Control Groups (continued)	176
4.9	Descriptive Statistics for the Experimental and Control Groups (continued)	178
4.10	Neuropathic Pain Results	186
4.11	Mean Scores of Experimental Group Compared to Patient A at Pre-test	197
4.12	Mean Scores of Experimental Group Compared to Patient A at Post-test	197
4.13	Mean Scores of Control Group Compared to Patient B at Pre-test	198
4.14	Mean Scores of Control Group Compared to Patient B at Post-test	199
4.15	Comparison of Post-Test scores for Patient A and Patient B	200

## LIST OF FIGURES

<b>Figure</b>	<b>Page #</b>
2.1 The Nociceptive Pain System	22
2.2 A Radiological View of the Spine	49
2.3 The Lumbar Vertebrae	50
2.4 The Intervertebral Disc Between Two Adjacent Vertebrae	52
2.5 The Structure of an Intervertebral Disc	54
2.6 The Composition of the Intervertebral Disc Structures	55
2.7 The Muscles of the Spine	62
3.1 The Straight Leg Raise Test	130
3.2 The International Standard Protractor Goniometer	130
3.3 The Slump Test	132
3.4 The Sorensen Back Extension Test	134
3.5 The Ito Test	135
3.6 The Side Bridging Endurance Test	137
3.7 The Flexor Endurance Test	137
4.1 Difference Between Experimental and Control Groups on Pre-test Measurements	187
4.2 Difference Between Experimental and Control Groups on Pre-test Measurements (continued)	187
4.3 Difference Between Experimental and Control Groups on Pre-test Measurements (continued)	188
4.4 Difference Between Experimental and Control Groups on Pre-test Measurements (continued)	188
4.5 Difference Between Experimental and Control Groups on Post-test Measurements	189
4.6 Difference Between Experimental and Control Groups on Post-test Measurements (continued)	190
4.7 Difference Between Experimental and Control Groups on Post-test Measurements (continued)	190



4.8	Difference Between Pre-test and Post-test Measurements within the Experimental Group	192
4.9	Difference Between Pre-test and Post-test Measurements within the Experimental Group (continued)	192
4.10	Difference Between Pre-test and Post-test Measurements within the Experimental Group (continued)	193
4.11	Difference Between Pre-test and Post-test Measurements within the Control Group	194
4.12	Difference Between Pre-test and Post-test Measurements within the Control Group (continued)	195
4.13	Difference Between Pre-test and Post-test Measurements within the Control Group (continued)	195

## LIST OF EXERCISE PHOTOS

<b>Exercise Illustration</b>	<b>Page #</b>
<b>Control Group</b>	
Cycling	138
Both Knees to Chest Stretch	139
Hamstring Stretch	139
Periformis Stretch	139
Roll Both Knees to Side	139
Sit on Stability Ball	140
Alt Superman on All-fours	140
Hip Lifts (Feet Flat on Floor)	140
Prone Alt Leg Lifts	140
Prone Alt Arm and Leg Lifts	141
<b>Experimental Group Programme 1</b>	
Cycling	142
Hamstring Stretch with Foot Flexion	142
Side Lying Quadricep Stretch	142
Lat Pulldown to the Front	142
Side Bridging (on Knees)	143
High Cable Horizontal Adduction (Downwards)	143
Hip Lifts with Feet on Bench	143
Alt Superman on Stability Ball	143
Abdominal Crunches (Feet on Bench)	144
<b>Experimental Group Programme 2</b>	
Cycling	144
Hamstring Stretch with Step-off	145
Side Lying Quadriceps Stretch	145
Lat Pulldown to Front	145
One-arm Dumbbell Row	146
Side Bridging (on Feet)	146

Low Cable Shoulder Flexion (Straight Arm)	146
Ball Squat Against Wall	147
Hip Lifts (Feet on Ball)	147
Alt Superman (Sweeping Hand on Floor Upon Return and Up Again)	147
Abdominal Crunches (Feet on Stability Ball)	148

### **Experimental Group Programme 3**

Cycling	148
Periformis Stretch	148
Rotation Stretch	148
Side Lying Quadriceps Stretch	149
Lat Pulldown to Front	149
High Cable Pulldown to Opposite Hip with Both Arms	149
Seated Cable Row	149
Ball Squat Against Wall (With Weight)	150
Side Bridging (on Feet, Lift Side)	150
Hip Lifts With One Leg at a Time (Feet on Bench)	150
Alt Superman	151
Abdominal Crunches (Lying on Ball)	151

## LIST OF ABBREVIATIONS

ACSM	American College of Sport Medicine
Alt	Alternative
BMI	Body Mass Index
cm	Centimetres
FABQ	Fear Avoidance Beliefs Questionnaire
FRI	Functional Rating Index
FWCA	Full Working Capacity Adults
Hrs	Hours
kg	Kilogram
min	Minute(s)
MVC	Maximal Voluntary Contraction
reps	Repetitions
RPE	Rate of Perceived Exertion
sec	Seconds
SIJ	Sacro-iliac Joint
SRL	Straight Leg Raise
VAS	Visual Analog Scale

## CHAPTER 1

### THE PROBLEM

#### 1.1 Introduction

Traditionally, the most diagnosed characteristic of low back disorders is pain, which has become a very common disorder (Goldby *et al.*, 2006). In recent times, pain has been recognized as a disease in itself with its own set of consequences, and no longer just as a symptom of some other type of disease or disorder (Meyer, 2007).

All innervated spinal tissues can be a potential source of pain, and due to the multifactorial etiology of chronic low back problems it has been suggested that there is still a large amount of uncertainty to the cause of low back pain (Kääpä *et al.*, 2006). Low back problems can arise from area of the extensive network of intersecting nerve fibres that supply the lumbosacral region, the vertebral periostium, intervertebral discs, neurovascular region, back extensor muscles, tendons, ligaments, vessels, fascias, zygapophyseal joints and sacroiliac joints, or it can even arise from the visceral organs (Schwarzer *et al.*, 1994; Freemont *et al.*, 1997; Simmonds & Dreisinger, 2003; Adams, 2004).

When applied load exceeds the failure tolerance, injury or tissue failure can result, which includes injuries from micro-trauma right up to total failure of the tissue such as ligament avulsion accompanying a fracture (McGill, 2002). The human pain experience is a multidimensional feature with sensory-discriminative, affective-motivational motor and autonomic components, which is critical to the survival of human beings and to maintain the integrity of the body. Pain has been described as an emotional, subjective and complex sensation (Treede *et al.*, 1999).

Pain is not always an indication of underlying problems. There exists a lack of understanding with regard to the multifactorial nature of low back problems, and this is reflected in the large variety of available treatments, ranging from medically oriented treatments such as injection therapy and surgery to behaviour oriented approaches (Nelemans *et al.*, 2001; Van Tulder *et al.*, 2001; Staal *et al.*, 2005). A large variety of treatments exists for the treatment of low back pain, from invasive procedures such as surgery and injection therapy to more psychology-based

approaches. Due to this, it has been suggested that there is a lack of understanding with regards to the multifactorial nature of low back pain. It has also been suggested that pain is not always an indicator of underlying problems (Nelemans *et al.*, 2001; Van Tulder *et al.*, 2001; Staal *et al.*, 2005).

Low back pain has a prevalence rate of about 84% (Simmonds & Dreisinger, 2003; Hildebrandt *et al.*, 2004). A prevalence rate of about 63.9% has been reported in South Africa (Van Vuuren *et al.*, 2005). About 44-78% of people suffer relapses in low back pain after an initial episode and 26-37% experience relapses in absence from work (Hildebrandt *et al.*, 2004). In those suffering from low back pain, about 11-12% are disabled by their pain.

In all cases of low back pain, being able to trace the pain back to a specific cause is rare, with only 15% of all cases able to be traced back to a specific cause (Hildebrandt *et al.*, 2004). Chronic low back pain will be the end result for about 5-10% of those who suffer an initial episode of back pain (Linton *et al.*, 2005). These patients undergo considerable suffering because of pain and reduced function as well as accounting for high costs from health care utilization and compensation for lost work (Dionne, 1997).

Direct and indirect costs of low back pain in the United States annually has been reported to be around \$25-\$50 billion because of pain and disability, which continues to rise and contributes to a substantial economic burden (Frymoyer & Cats-Baril, 1991; Frymoyer, 1992). The losses due to loss of productivity are around \$28 billion (Rizzo *et al.*, 1998). Those with low back pain also suffer higher health care costs, which are around 60% greater than healthy individuals (Luo *et al.*, 2004).

This again demonstrates the severe impact of low back pain on society. Primary health care consultation in the United Kingdom for low back pain is the second highest reason why health care is sought (Deyo & Phillips, 1996). Medical expenditure in the United States due to low back pain is responsible for billions of dollars being spent each year (Childs *et al.*, 2004). This impact of low back pain on the economy is of particular concern for poorer continents such as Africa, where epidemics such as HIV/AIDS already take up a majority of the governmental

expenditure towards health care (Walker, 2000). This again demonstrates the severe impact of low back pain on society.

Work absenteeism accounts for a large proportion of the socio-economic burden of low back pain. Restrictions on normal activities and the participation in activities such as the ability to work can lead to absenteeism. This can be caused by low back pain that leads to disability, which will place significant restrictions on these usual activities (Katz, 2006). The largest number of low back pain related economic costs in Western societies is reported to include work absenteeism and disablement (Andersson, 1999). It has also been reported that the majority of low back pain episodes will still result in a full return to work capacity (Phelps *et al.*, 2004), but those that become chronic has been widely reported to be responsible for a large portion of the total number of work absenteeism and accounts for a large portion of the economic and social burden of low back pain (Burton, 2005).

An important part of the treatment for low back pain has been suggested to be a return to work, which should be more than just a treatment goal and an outcome measure that is used in research (Staal *et al.*, 2005). When the disabled worker is involved in the work setting, they can realize that despite the discomfort due to pain that they can still function. Being at work, either in partial or full-capacity, attention is drawn away from the negative issues such as pain and helps to decrease the focus of the disabled worker from pain (Staal *et al.*, 2005).

This demonstrates the need for those with chronic low back pain to remain active regardless of the discomfort suffered. A very important economic implications is that patients who are absent from work for more than six months has a 50% chance of returning to work. This number becomes lower the longer the person is away from work. Being off from work for more than one year has a 25% chance of returning to work and being off for longer than two years has less than a 5% chance of returning to work (Bergquist-Ullman & Larsson, 1977).

The summary of the recommendation comes down to the guidelines that recommend that when a person first represents with acute low back pain, they first have to be examined for the so-called 'red flags', which are indications of serious underlying

pathology (Koes *et al.*, 2001). If the patient doesn't present with any red flags, current recommendations state that they should be advised to continue or gradually resume their activities of daily living (Waddell *et al.*, 1997; Koes *et al.*, 2001; Waddell, 2004). Beyond this, further recommendations state that treatment has to be delayed until the patient has been away from work for at least 4-6 weeks. This is only to prevent the slip into chronicity, as many patients will recover spontaneously from an episode of acute low back pain (Frank *et al.*, 1996).

Psychological, social and occupation factors increase in influence of back pain becoming chronic, because the longer that a patient experiences low back pain and subsequent disability, the more the factors will become significant (Waddell *et al.*, 1996). For the treatment of chronic low back pain, conservative treatment has become the most recommended form of treatment (Shirado *et al.*, 2005). Absence from work because of back pain recommended to be prevented by physical exercise, as well as preventing further episodes and the severity of pain episodes (Burton, 2005). Exercise therapy is thus recommended by many guidelines, and it is one of most recommended guidelines for the treatment of chronic low back pain (Spitzer *et al.*, 1987; Albright, 2001; Hayden *et al.*, 2005; Krismer & Van Tulder, 2007). The pathogenesis of low back pain shares a close relationship with impaired function of the trunk muscles (Shirado *et al.*, 1992; Shirado *et al.*, 1995a; Ito *et al.*, 1996).

To strengthen the trunk muscles and improving their flexibility is the primary purpose of therapeutic exercises for chronic low back pain (Shirado *et al.*, 1995b). It is recommended by current evidence to use multidisciplinary/interdisciplinary approaches in the treatment for chronic low back pain, because multiple therapies are incorporated in a coordinated manner which facilitates active interaction and a common philosophy that promotes active involvement from the patient in the rehabilitation programme (Ashburn & Rice, 1998; Ashburn & Staats, 1999; Karjalainen *et al.*, 2001; Karjalainen *et al.*, 2003a; Rome *et al.*, 2004).

This approach includes either one of the psychological, social or occupation dimensions along with the physical exercise therapy (Guzman *et al.*, 2001). Functional capacity and the development of coping strategies was the reason that the multidisciplinary programmes have been developed (Linton & Andersson, 2000).



Providing accurate information about back pain, to lend attitudes that are favourable towards self-care, to reduce fears and worries, to assist patients in developing personalized action plans to manage their back pain and to improve functional outcomes are all established goals of the multidisciplinary approach (Kääpä *et al.*, 2006).

The use of exercise therapy as part of the multidisciplinary approach has become very popular for the treatment for chronic low back pain (Staal *et al.*, 2005). However, as suggested by McGill (2002), many of the studies focusing on exercise treatments have not made assessments about progressive treatment that change as the patient move through the rehabilitation process, and have only focused on single treatment methods.

Patient needs change throughout the rehabilitation process. Therefore, the applied exercises should also change and adapt to accommodate these changes by increasing in difficulty as the levels of exercise tolerance of the subject increase (McGill, 2002). Also, many of the exercise programmes have been conservative in nature (Richardson *et al.*, 1999; Hides *et al.*, 2001) and it is not clear if this is sufficient to restore full functional capacity.

The important part will be to select exercises that are safe, yet effective. In the rehabilitation process, functional progression is needed to stress tissue sufficiently that will allow it to adapt to other kinds of stresses that a patient will be exposed to in every situations. Otherwise, tissue failure due to weakness can lead to injury (Prentice, 2004). Unfortunately, the effect of more aggressive types of exercises has not been assessed adequately. In order to fully restore functional capacity and provide tissue with enough strength to sustain loads applied to the body, it might be necessary to include more aggressive types of exercises to not only strengthen the muscles of the low back, but also to strengthen the muscles used for functional tasks in a safe and effective way (McGill, 2002). The important part will be to select exercises that are safe, yet effective.

Approaches that put emphasis on functional restoration and the improvement of functional capacity produces the best results in the management of chronic low back

pain. These approaches focus on the development of coping strategies to help those with chronic low back pain in the long term (Linton *et al.*, 2000; Caraggee, 2005). It thus provides them with a type of self-management strategy (Rasmussen-Barr *et al.*, 2009).

Taking this into account, it can be argued that treating musculoskeletal dysfunctions alone may not be beneficial without directly addressing psychosocial factors that contribute to the experience of pain (Geisser *et al.*, 2005), thus combining more than one treatment modality. This is especially true if the type of pain is considered in chronic low back pain, which does not always indicate that a problem exists (Meyer, 2007).

Several recent studies have provided evidence that treatment programmes containing active exercises are equally effective in patients with chronic low back pain, irrespective of the type of exercises compared. This suggests that any type of intensive exercise programme that manages to cause patients to expand the limits of their physical functioning may provide them with a method for increasing the feeling of pain control, thus inhibiting negative pain behaviour relating to a chronic low back problem (Petersen *et al.*, 2002).

In a well-designed randomised controlled trial Petersen *et al.* (2002) compared the effect of McKenzie therapy to intensive strengthening in the treatment of chronic low back pain. They concluded that the McKenzie method and intensive strength training seem to be equally effective in the treatment of chronic low back pain. However, frequently used, popular exercise regimens, like the McKenzie technique, are not adequately researched.

The European Guidelines for the Management of Chronic Non-Specific Low Back Pain suggest that there is a need to investigate the optimal intensity, frequency, duration and specific types of exercises used in the clinical setting and academic literature (Hildebrandt *et al.*, 2004). It also remains unclear whether any specific type of exercise (flexion, extension or strengthening exercises) is more effective than another (Van Tulder *et al.*, 2000).

Problems associated with chronic low back pain such as restricted functioning, disability and absence from work can cause more long-term problems and disability than pain alone, and it has been suggested that pain treatment has to be a secondary goal, as these other problems cause more long-term complications (Staal *et al.*, 2003; Staal *et al.*, 2005). Treatment should thus focus rather on the consequence of pain, such as a loss of function, physical inactivity and work absenteeism. Pain varies from day to day and is thus not necessarily of long-term concern (Staal *et al.*, 2005). A high recurrence rate of low back pain will likely lead to a life that is not totally free of pain, but pain can be managed and restricted (Staal *et al.*, 2005).

Future studies must assess the efficacy of staged programmes in which categorised patients follow progressive treatment involving several sequenced approaches (McGill, 2002). More aggressive types of remedial exercises, as well as specific types of exercises have to be incorporated in order to establish if they are more effective in the treatment of chronic low back pain.

## 1.2 Research Questions

For this study the following research questions were used:

- ***How effective are progressive-aggressive exercises vs. more traditional exercise in the treatment of chronic non-specific low back pain?***
- ***What exercises will be effective and how effective they will be when progressed?***
- ***How will a more aggressive approach influence the outcomes as compared to the more traditional approaches to remedial exercise therapy?***

## 1.3 Research Hypothesis

In the light of the aim of this study the following research hypothesis was formulated:

***A progressive-aggressive multi-component rehabilitation programme in conjunction with high intensity back school will be more effective in the***

***improvement of chronic low back pain outcomes than a conservative rehabilitation programme in conjunction with a low intensity back school.***

Significance was set at  $p \leq 0.01$  level of significance.

#### **1.4 Goals of the Study**

The following goals were set prior to the study commencing:

***Develop a progressive-aggressive exercise programme, a conservative exercise programme and a back school curriculum.***

#### **1.5 Objectives of the Study**

This study aimed to achieve the goal through the following objectives:

- Building a theoretical frame of reference on existing literature with specific focus on topics such as chronic pain, work absenteeism, remedial exercise therapy and full working capacity adults;
- Identification of the major components involved in chronic low back pain, as well as the mechanisms involved in chronic pain (neuropathic pain, etc.);
- Description of the remedial exercise use and function associated with chronic low back pain;
- Identification and description of a test battery that will be suitable in assessing the critical components of the low back;
- Description and illustration of the different exercises to be used with the progressive-aggressive exercise programmes, as well as the conservative exercise programmes;
- Guidelines for the implementation of the progressive-aggressive exercise programme; and
- Suggestions for future research on the topic of low back pain, which remains a topic with many unanswered questions.

## 1.6 Research Design

The present study mainly made use of qualitative research methods. Qualitative research methods generally include field observations, case studies, ethnography and narrative reports (Linn, 1986). This type of research seeks to understand the meaning an experience has for the participants in a specific setting and how the components mesh to form a whole (Thomas & Nelson, 2001).

The objectives are primary description, understanding and meaning. Thus the researcher does not manipulate the variables through experimental treatments alone but rather takes more interest in the process than the product (Patton, 1987). The researcher observes and gathers data in the field, which is the natural setting (Thomas & Nelson, 2001). Qualitative research methods, however, are very subjective. Here the researcher is the primary instrument for data collection and analysis, as well as interacting with the subjects to observe their responses and changes (Patton, 1987).

The type of research design used in the present study was that of experimental research. With this type of research the independent variables are manipulated in an attempt to judge their effect on the dependant variable. A well-designed study is one in which the only explanation for the change in the dependant variable will be due to the applied intervention (Thomas & Nelson, 2001). Thus, the major advantage of this type of research is that the researcher is able to manipulate the treatments to establish a cause-and-effect relationship (Thomas & Nelson, 2001). Cause and effect can only be established by the application of logical thinking to well-designed experiments (Kratwohl, 1993).

Three criteria need to be present to establish cause and effect:

1. The cause must precede the effect in time.
2. The cause and effect must be correlated with each other.
3. The correlation between cause and effect cannot be explained by another variable. (Kratwohl, 1993)

The researcher attempts to control all factors except the experimental variable. If extraneous factors can be controlled successfully, then the researcher may presume that the changes in the dependant variable are due to the independent variable (Thomas & Nelson, 2001).

This presumption has to include the following aspects:

- The selection of a good theoretical framework
- The use of appropriate participants
- The application of an appropriate experimental design
- The proper selection and control of the independent variable
- The appropriate selection and measurement of the dependent variable
- The use of the correct statistical model and analysis
- The correct interpretation of the results (Kratwohl, 1993)

The type of study design used in the present study was a True Experimental Design, which basically facilitates the random allocation of groups to ensure that all sources of invalidity and all threats to design have been counteracted (Thomas & Nelson, 2001). More specifically, the present study was a Pre-test-post-test Randomised Groups Design, in which the groups were randomly formed but both groups were also given a pre-test, as well as a post-test. The major purpose of this type of design is to determine whether the experimental group showed more improvement than the control group (Thomas & Nelson, 2001).

## **1.7 Research Procedure and Strategy**

This study included subjects, both male and female, between the ages of 20 and 55 years of age. They were randomly assigned to either a control or experimental group. Each participant was also assigned a unique study number and this number will be randomly linked to the data. Patients' names and file numbers will have no correlation to the study number. The unique study number will prevent patients from

being identified, even by the researcher, and this will ensure the confidentiality of the records in order to protect patients' privacy.

### **1.7.1 Inclusion Criteria**

Inclusion criteria consisted of suffering from back pain for longer than 12 weeks, based on the classification of chronic low back pain recommended by Abenheim *et al.* (2000) and Burton *et al.* (2004). The source had to be chronic in nature, which was confirmed by the physical testing procedures and questionnaires. Subjects could not suffer from low back pain caused by the so-called 'red flag' conditions. Subjects were included who suffered from radiating symptoms in the legs, but this again could not be caused by any 'red flag' conditions.

### **1.7.2 Exclusion Criteria**

Exclusion criteria consisted of any previous back surgery, any known spinal pathology and discogenic diseases, as well as the so-called 'red flag' conditions. These include weakness, particularly if localised in one area such as the leg; pain and/or difficulty controlling the bladder; numbness or tingling in the feet, legs or groin; severe, disabling or night pain; serious pain and a history of cancer or intravenous drug use; pain that does not subside within a couple of days; pain in the abdomen, as well as fever and weight loss along with back pain.

Current pregnancy, as well as ongoing disability and injury compensation cases were also excluded. Subjects were not excluded based on previous treatment modalities such as physiotherapy. However, they could not seek these types of treatments during the course of the study. Subjects were also excluded if they had a Body Mass Index (BMI) of over 40. By default this condition puts too many compressive forces on the low back and may contribute to chronic low back pain (McGill, 2002).

### **1.7.3 Study Sample**

Subjects for this study were recruited by placing advertisements in local newspapers, as well as broadcasting the advertisement on the local radio station. Physicians were also contacted for possible referrals for likely and willing participants seeking long-term help. After initial contact with the possible subjects, they underwent a thorough

screening procedure by the main investigator to ensure that they fitted the criteria of chronic low back pain. Potential subjects then gave written informed consent prior to participation. The subjects selected were then randomly assigned to either the control or the experimental group.

Research subjects were given the questionnaires to complete at the initial testing session. After the 12 week intervention period they were again given the questionnaires to complete. Subjects were given 20 minutes to complete the questionnaires. The researcher administering the tests was blinded to the allocation of the subjects at each of the test sessions by means of random numbers. Immediately after the questionnaires had been completed, the physical tests were administered on the subjects. The questionnaires were repeated after every four weeks during the intervention period when the exercise programme of the experimental group progressed.

To prevent test results from being confused, the subjects were tested individually and not allowed to interact with one another during the test administration. This prevented subjects from copying one another's answers because of what they thought the answers should be as required by the researchers. Subjects had to provide answers to what they were experiencing; not to what they thought the researchers wanted to hear.

#### **1.7.4. Intervention**

- The timeframe for the intervention was 12 weeks.
- Subjects were randomly assigned to two groups. The control group performed the conservative exercise programme while the experimental group performed the progressive-aggressive exercise programme.
- After every four weeks the exercise programme of the experimental group was progressed to the next level of difficulty (at Week 4 and Week 8 up to the maximum of Week 12).
- Both groups performed the exercise sessions twice per week.



- The control group also received low intensity back school intervention while the experimental group received high intensity back school intervention.
- High intensity back school consisted of two sessions per week for 40 minutes, where the principles of the cognitive behavioural approach was applied. This included the exercise session and the back school session.
- In turn, the control group performed the low intensity back school in the form of reading the back school document on their own, but still exercised twice per week. The control group back school was done in the form an informative book given to the subjects to study. This procedure is based on the guidelines suggested by Heymans *et al.* (2006).

### 1.7.5 Back School

The back school included a high intensity approach for the experimental group and low intensity for the control group. High intensity-based back schools have been shown to be more effective to a certain degree than low intensity back schools (Heymans *et al.*, 2006). High intensity back school consisted of two sessions per week for an hour (including the exercise session) during which the principles of the cognitive behavioural approach were applied.

In turn, the low intensity back school was applied twice a week (with two exercise sessions per week) during which subjects were handed an informative book. This book contained all of the necessary information but subjects had to study the content on their own. The experimental group was given information as well but it was discussed with them in detail. This procedure is based on the guidelines suggested by Heymans *et al.* (2006).

## 1.8 Definition of Key Concepts

- **Back Pain:** Pain and discomfort localised below the costal margin and above the inferior gluteal folds, with or without leg pain (Van Tulder *et al.*, 2004)

- **Pain:** An unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage (Meyer, 2007).
- **Chronic Low Back Pain:** Low back pain that persists for more than 12 weeks (Burton *et al.*, 2004). This is pain that persists for longer than the time expected for healing or pain associated with progressive, non-malignant disease, usually taken to be three months (Meyer, 2007).
- **Therapeutic Exercise:** Exercise performed for the purpose of cure or restoration of function (Abenham *et al.*, 2000).
- **Progressive-Aggressive Exercise:** Exercise that increases in difficulty as the needs of the patient change to create a need for higher levels of intensity and exercise tolerance (McGill, 2002).

For the purpose of this study, conservative exercise will imply exercises that aim to strengthen the local muscle group only. Aggressive exercises will include exercises that strengthen the local muscle group as well as the global muscle group.

- **Full Working Capacity Adults:** Adults who suffer from chronic low back pain but suffer virtually no disability at all due to the pain. They are still working full-time in terms of hours, duties and travelling time, but are required to take part in a back rehabilitation programme.
- **Multidisciplinary/Interdisciplinary Rehabilitation:** Rehabilitation which includes the physical dimension and at least one of the other dimensions: psychological, social or occupational dimensions (Guzman *et al.*, 2001).
- **Neuropathic Pain:** Injury or disease of neurons in the peripheral or central nervous system (Jänig & Baron, 2003).
- **Fear Avoidance Behaviour:** The perception of pain in a threatening, catastrophic manner (as in signs of tissue damage); experiencing of pain-

related fear and anxiety, and consequently engaging in escape or avoidance behaviours to situations that are perceived to be potentially harmful (Thomas & France, 2007).

## CHAPTER 2

### LITERATURE SURVEY

#### 2.1 Pain and its Physiology

Pain is an important symptom of a wide variety of different diseases and disabilities (Schaible & Richter, 2004). However, as stated by Meyer (2007), chronic pain has been recognised in recent times as not only a symptom, but also as a disease itself.

According to Meyer (2007:20), a current definition of pain proposed by the IASP (International Association for the Study of Pain) states: "*Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage*". The casual mechanism in pain, especially musculoskeletal pain is a very complex phenomenon, as the experience of pain itself may emphasize the negative effects of experienced external stressors, or provoke psychological and biological reactions that maintain or increase pain in a vicious circle (Svebak, 2000).

It has been reported that the experience and regulation of social and physical pain share a common neuro-anatomical basis in the brain (Eisenberger *et al.*, 2003). Pain can be described as a sensation that is evoked by potential or actual noxious (toxic) stimuli or by actual tissue damage (Schaible & Richter, 2004). A potential source of low back pain for example can arise from any part of the extensive network of intersecting nerve fibres that supply the lumbosacral region, the vertebral periostium, intervertebral discs, neurovascular region, back extensor muscles, tendons, ligaments, vessels, fascias, zygapophyseal joints and sacroiliac joints, or it can even arise from the visceral organs (Schwarzer *et al.*, 1994; Freemont *et al.*, 1997; Simmonds & Dreisinger, 2003; Adams, 2004).

Also, advanced atherosclerosis presenting calcific deposits on the posterior wall of the abdominal aorta and in vertebral arteries has been suggested to be associated with advance types of disc degeneration and the subsequence occurrence of low back pain (Kauppila *et al.*, 1997).

Injury or tissue failure can also occur when the applied load exceeds the failure tolerance of the muscle, and includes micro-trauma all the way up to gross tissue failure, such as fractures and ligament avulsion (McGill, 2002). However, it is generally accepted that non-innervated tissue cannot be the origin of pain sensation (Aoki *et al.*, 2006), such as the intact lumbar intervertebral disc, which has been shown to be aneural except in the outermost part of the annulus fibrosus (Bogduk *et al.*, 1981; Coppes *et al.*, 1990; Konttinen *et al.*, 1990; Roberts *et al.*, 1995; Freemont *et al.*, 1997; Palmgren *et al.*, 1999).

These nerves extend no further than the outer few lamellae of the annulus fibrosis (Coppes *et al.*, 1990; McCarthy *et al.*, 1991; Freemont *et al.*, 1997). Some of these nerves contain neurotransmitters such as substance P and calcitonin gene-related peptide, indicating a possible nociceptive function and subsequent pain regulation function (Ahmed *et al.*, 1991; Grönblad *et al.*, 1991; McCarthy *et al.*, 1991; Ahmed *et al.*, 1993; Ashton *et al.*, 1994; Roberts *et al.*, 1995).

However, both nerve fibre and blood vessel ingrowth deeper into the annulus fibrosis and even up to the nucleus pulposus have been demonstrated in degenerative discs, acting as a possible source of pain perception (Coppes *et al.*, 1990; Kauppila, 1995; Palmgren *et al.*, 1996; Freemont *et al.*, 1997; Repanti *et al.*, 1998). Even high levels of serum blood cholesterol levels have been associated with low back pain in some people (Kauppila *et al.*, 2004).

This network of tissue is the reason why the source of pain is frequently difficult to determine, as any innervated structure of the low back can trigger a possible nociceptor and thus a painful signal, and most of the structures in the low back are well innervated, relatively small and in close proximity to each other (Simmonds & Dreisinger, 2003).

Pain can be classified as being either acute or chronic in nature (Meyer, 2007). Acute pain is usually of known cause, and there are only minor contributions from the emotional and cognitive dimensions, but in chronic pain patients the pain can last for up to many years, and the emotional and cognitive dimensions can potentially play a much larger role in pain perception over a long period of time (Johnson, 1997;

Simmonds & Dreisinger, 2003). According to Meyer (2007) a large number of brain regions that are also described as the so-called “pain matrix” are activated following a stimulus that causes pain. Rather than registering the pain signal, it is communicated to the body as a perception of pain, and the brain matrix constructs the pain experience by integrating multiple inputs that includes biological (sensory) factors, past and present psychological events and socio-cultural influences.

Recognizing the importance of pain in modern research settings, Schaible & Richter (2004: 237) stated the following: “*Pain is a major symptom of many different diseases. Modern pain research has uncovered important neuronal mechanisms that are underlying clinically relevant pain states, and research goes on to define different types of pains on the basis of their neuronal and molecular mechanisms. This review will briefly outline neuronal mechanisms of pathophysiological nociceptive pain resulting from inflammation and injury, and neuropathic pain resulting from nerve damage.*”

The human pain experience is a complex sensation that is of paramount importance to maintain the body integrity and survival of human beings, as it is a multidimensional phenomenon with sensory-discriminative, affective-motivational, motor and autonomic components (Treede *et al.*, 1999). Treede *et al.* (1999) argued that the human pain experience is a complex sensation that is of paramount importance to maintain the body integrity and survival of human beings. It is a multidimensional phenomenon with sensory-discriminative, affective-motivational, motor and autonomic components, making it a very effective phenomenon. Understanding the function of the pain system is of primary concern.

### **2.1.1 The Pain System**

Schaible & Richter (2004: 237) stated that:” *Precisely, the ‘pain system’ should be called the ‘nociceptive system’ because pain is a subjective result of nociception. Nociception is the encoding and processing of noxious stimuli in the nervous system that can be measured with electrophysiological techniques.*” This results due to the activation of complex and integrated networks of neurons that are prone to string loops consisting of automatic regulation and fast changing neuroplastic responses (Verdu *et al.*, 2008).

Schaible & Richter (2004: 237) further stated that:” *A noxious stimulus activates nociceptors (A $\alpha$  and C fibres) in the peripheral nerve. Their sensory endings are so-called free nerve endings, i.e. they are not equipped with corpuscular end organs. Most of the nociceptors are polymodal, responding to noxious mechanical stimuli (painful pressure, squeezing or cutting of the tissue), noxious thermal stimuli (heat or cold), and chemical stimuli (Belmonte & Cervero, 1996).*” This opens ion channel transducers to accelerate the nociceptive pain experience (Verdu *et al.*, 2008).

Schaible & Richter (2004: 237) further states that: ”*Sensor molecules in the sensory endings of nociceptors transduce mechanical, thermal and chemical stimuli into a sensor potential, and when the amplitude of the sensor potential is sufficiently high, action potentials are triggered and conducted by the axon to the dorsal horn of the spinal cord or the brainstem. Nociceptors can also exert efferent functions in the tissue by releasing neuropeptides [substance P, calcitonin gene-related peptide (CGRP)] from their sensory endings. Thereby, they induce vasodilatation, plasma extravasation and other effects, e.g. attraction of macrophages or degranulation of mast cells. The inflammation produced by nociceptors is called neurogenic inflammation (Foreman, 1987; Lynn, 1996).*”

Schaible & Richter (2004: 237-238) further states that: “*Nociceptors activate synaptically nociceptive dorsal horn neurons. The latter are either ascending tract neurons or interneurons that are part of segmental motor or vegetative reflex pathways. Ascending axons in the spinothalamic tract activate the thalamocortical system that produces the conscious pain sensation. The pain sensation has a sensory discriminative aspect, i.e. the noxious stimulus is analysed for its location, duration and intensity. This is produced in the lateral thalamocortical system, which consists of relay nuclei in the lateral thalamus and the areas SI and SII in the postcentral gyrus. A second component of the pain sensation is the affective aspect, i.e. the noxious stimulus feels unpleasant and causes aversive reactions. This component is produced in the medial thalamocortical system, which consists of relay nuclei in the central and medial thalamus and the anterior cingulate cortex (ACC), the insula, and the prefrontal cortex (Basbaum & Jessell, 1999; Treede *et al.*, 1999).*

*The spinal cord is under the influence of descending tracts that reduce or facilitate the nociceptive processing. Descending inhibition is formed by pathways that originate from brainstem nuclei (in particular, the periaqueductal grey, nucleus raphe magnus) and descend in the dorsolateral funiculus of the spinal cord. This system is able to suppress nociceptive information processing via interneurons in the dorsal horn of the spinal cord (Basbaum & Jessell, 1999)”.*

However, the concept of the nociceptive pain system being the only cause of physiological pain production has been challenged by the so-called ‘gate-control theory’ of Melzack and Wall in 1965, which integrates the views of neurophysiology and psychology (Melzack & Wall, 1965).

According to Melzack & Wall (1965), the theory states that spinal transmission of pain impulses is continuously modulated by the relative activity in the small (A-delta and C) fibres and the large (A-beta) fibres. The descending impulses from the brain that originate in the cerebral cortex and brainstem also plays a role, and the subsequent irritation that is felt as pain will be reinforced by different parts of the central nervous system.

Under these circumstances, psychological and social factors also have an influence on pain sensation (Nykänen & Koivisto, 2004). Revolutionary was the finding that pain is not just the result of nociceptive information ascending from the periphery, but that it is also profoundly moderated by descending pathways (Vlaeyen & Morley, 2005). It is because of the advent of this theory that the modern paradigm of pain management has moved away from the classic biomedical approach to the broader bio-psycho-social approach, where pain physiology now integrate input from sensory, emotional and cognitive systems (Main & William, 2002; Merskey *et al.*, 2005; Justins, 2005).

The biomedical perspective has thus evolved into a broader conceptual framework, which addresses the influence and effect of psychological and social factors in the pathophysiology and prognosis of pain and subsequent disability (Staal *et al.*, 2005). Moreover, the nociceptive signal is modified during its transmission from the



peripheral nerves to neurons in the spinal cord and then up to the sensory centres of the brain.

The interpretation of the pain signal is now recognized to be influenced by numerous psychological (such as past experience and mood) and social (such as work, leisure activity) factors (Simmonds & Dreisinger, 2003). This change in thinking identifies the complex and multi-dimensional experience of pain where the patient's physical, cognitive, emotional and behavioural characteristics mediate the pain experience and can influence the reaction of the person based on these factors (Katz, 2000). Currently, chronic pain states are attributed to abnormal nociceptive/antinociceptive function on different levels of the neuroaxis with normal brain structure (Wall & Melzack, 1999).

### **2.1.2 Types of Pain**

Different types of pain can be distinguished. On the bases of aetiology and neurobiological mechanisms Treede *et al.* (2008) have identified different types of pain:

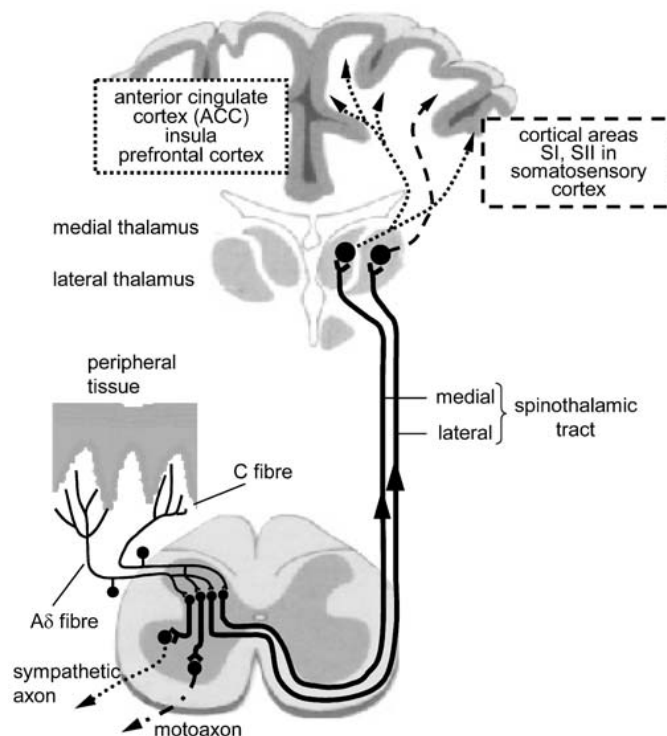
- 1) nociceptive pain, caused by any lesion or potential tissue damage;
- 2) inflammatory pain, due to inflammatory processes; and
- 3) neuropathic pain, induced by a lesion or disease affecting the somatosensory system (Treede *et al.*, 2008).

In the absence of a neurological disorder or peripheral tissue abnormality another type of pain has been suggested in the form of functional/dysfunctional pain, which is supported by the existence of an abnormal central operation of inputs leading to pain hypersensitivity (Talley & Spiller, 2002; Desmeules *et al.*, 2003; Banic *et al.*, 2004; Harris *et al.*, 2007).

#### **2.1.2.1 Nociceptive Pain**

Schaible & Richter (2004: 238) stated that: "*When a noxious stimulus is applied to normal tissue, acute physiological nociceptive pain is elicited. This pain protects tissue from being further damaged, because usually withdrawal reflexes are elicited. Pathophysiological nociceptive pain occurs when the tissue is inflamed or injured.*

*This pain may appear as spontaneous pain (pain in the absence of any intentional stimulation) and/or as hyperalgesia and/or allodynia.”*



**Figure 2.1:** The Nociceptive Pain System (Schaible & Richter, 2004)

Seifert and Maihöfner (2008) reported that hyperalgesia can be differentiated into primary and secondary hyperalgesia, where primary hyperalgesia results from the sensitisation of peripheral nociceptive structures, and secondary hyperalgesia results from sensitisation processes within the central nervous system. A hypersensitivity towards heat stimuli known as thermal hyperalgesia is a key component of primary hyperalgesia, whereas secondary hyperalgesia is recognised by hypersensitivity towards mechanical stimulation. Schaible & Richter (2004: 238) stated that:” *Hyperalgesia is a higher pain intensity that is felt upon noxious stimulation, and allodynia is the occurrence of pain that is elicited by stimuli that are normally below the pain threshold.*”

### 2.1.2.1.1 Peripheral Mechanisms of Pathophysiological Nociceptive Pain

The sensitizing of the polymodal nociceptors during inflammation is referred to as peripheral sensitization, and tend to have relatively high levels of mechanical and thermal thresholds in normal tissues. They require a high level of intensity from stimuli to cause an excitation of the involved neurons (Handwerker, 1999; Schaible & Schmidt, 2000).

This leads to a situation in which even very slight and non-significant stimuli activates the fibres due the excitation levels of the fibres decreasing during the inflammation process. This causes pain to be experienced due to the sensitized pain fibres which react to non-painful stimuli. Noxious stimuli will still cause a much stronger reaction when compared to the non-sensitized state (Handwerker, 1999; Schaible & Schmidt, 2000).

According to Schaible and Richter (2004) silent nociceptors can be activated by the inflammation process and these C-fibres are, under normal conditions, inexcitable by stimuli that are either thermal or mechanical. However, inflammation changes this condition, in that the primarily mechanosensitive fibres become sensitized and activate when a stimulus is applied. Thus the enhanced activity of the sensitized polymodal nociceptors and the subsequent recruitment of the silent nociceptors cause the pathophysiological nociceptive input to the spinal cord.

Interaction from inflammatory response mediators which include prostaglandins, bradykinin, histamine, ATP and acetylcholine on receptors on sensory endings (Kress & Reeh, 1996; McCleskey & Gold, 1999; Schaible, 2004) activates the neurons directly or causes a sensitization effect for other forms of stimuli (Kress & Reeh, 1996). The activation of secondary messenger by the mediators can then influence specific ion channels in the membrane. This type of reaction then causes an enhanced excitability of the neuron with decreased threshold levels and an increased action potential frequency during higher than normal activation (Bevan, 1996).

Schaible & Richter (2004:239-240) stated that: "*Primary afferent neurons also express receptors for neurotrophins. Neurotrophins are survival factors during the*

*development of the nervous system, but during inflammation of the tissue, the level of nerve growth factor (NGF) is substantially enhanced. By acting on the tyrosine kinase A (trk A) receptors, NGF increases the synthesis of substance P and CGRP in the primary afferents. The release of these peptides from the endings produces neurogenic inflammation. NGF may also act on mast cells and, thereby, activate and sensitise sensory endings by mast cell degranulation (Lewin et al., 1994). The sensitisation of nociceptors is rapidly induced, i.e. the changes mentioned can be observed within a few minutes. If noxious stimuli persist, changes in the expression of receptors in the primary afferents are induced. For example, the expression of neurokinin 1 receptors (activated by substance P) and bradykinin receptors is enhanced in rat dorsal root ganglia and in peripheral nerve fibres during persistent inflammation (Segond von Banchet et al., 2000)."*

#### **2.1.2.2 Neuropathic Pain**

Nociceptive pain has been described as pain that is caused by the stimulation of the sensory endings by noxious stimulation. Another type of pain known as neuropathic pain has also been described. This type of pain occurs due to an injury or a disease of the neurons specifically in the periphery or the central nervous system (Jänig & Baron, 2003). It can also include a lesion in the peripheral or central nervous system; for example, in patients with diabetic or AIDS poly-neuropathy and post herpetic neuralgia (Shipton, 1999; Woolf, 2004; Meyer, 2007).

Hyper-excitability and ectopic activity are unique to neuropathic pain, in that altered membrane excitability and abnormal electrogenesis results lead to the generation of inappropriate action potentials and repetitive firing without a peripheral stimulus (Verdu *et al.*, 2008). This type of pain doesn't cause normal noxious stimulation of the tissues and tends to feel like an abnormal type of pain, and has been described as a feeling of being burnt and/or a feeling similar to electricity passing through the area, which can become longstanding in nature, or occur in intermitted bursts and can even occur together with hyperalgesia and allodynia (Jänig & Baron, 2003). According to Verdu *et al.* (2008) hyper-excitability and ectopic activity is unique to neuropathic pain. Altered membrane excitability and abnormal electrogenesis result leads to the generation of inappropriate action potentials and repetitive firing without a peripheral stimulus

In the state of allodynia, sensitivity increases dramatically, so that even slight contact with the skin with non-painful touches will cause an intensive amount of pain (Schaible & Richter, 2004). Schaible & Richter (2004: 238) stated that “*Numerous pathological processes can cause neuropathic pain, e.g. axotomy or nerve or plexus damage, metabolic diseases such as diabetes mellitus, or herpes zoster. The complex regional pain syndrome (CRPS) is a neuropathic pain syndrome that involves the sympathetic nervous system (one form was previously called sympathetic reflex dystrophy or Sudeck’s disease) (Jänig & Baron, 2003). Damage to central pain processing neurons (e.g. in the thalamus) can cause central pain (Basbaum & Jessell, 1999).*”

#### **2.1.2.2.1 Peripheral Mechanisms of Neuropathic Pain**

Schaible & Richter (2004: 240) stated that: “*Ectopic discharges do not only occur in A $\alpha$ -fibres and C-fibres, but also in thick myelinated A $\beta$ -nerve fibres that encode innocuous mechanosensory information. This has led to the idea that, after nerve injury, low threshold A $\beta$ -fibres, as well as A $\alpha$ -fibres and C-fibres, are involved in the generation of pain. In particular, two mechanisms have been proposed as to how impaired A $\beta$ -nerve fibres might cause pain. First, A $\beta$ -fibres might evoke exaggerated responses in spinal cord neurons that have undergone the process of central sensitisation. Second, A $\beta$ -fibres might sprout into spinal cord layers that are usually only a target of C-fibres, and, thus, these fibres might activate the ‘wrong’ neurons (Woolf et al., 1992).*”

Schaible & Richter (2004: 240) further stated that: “*Different mechanisms are thought to produce ectopic discharges: changes in the expression of ionic channels, pathological activation of axons by inflammatory mediators, and pathological activation of injured nerve fibres by the sympathetic nervous system. At least six different types of sodium channels were found in primary afferents, two of them being tetrodotoxin (TTX)-insensitive (McCleskey & Gold, 1999). Sodium influx through TTX-sensitive sodium channels into the neuron inactivates very quickly: sodium influx through TTX-insensitive sodium channels is more slowly inactivating (Cummins et al., 2000). After nerve injury the expression of TTX-sensitive sodium channels is increased, and the expression of TTX-insensitive sodium channels is*

decreased, *These changes are thought to alter the membrane properties of the neuron, such that rapid firing rates (bursting ectopic discharges) are favoured (Cummins et al., 2000). Changes in the expression of potassium channels of the neurons have also been shown (Everill & Kocsis, 1999).*

*Injured axons of primary afferent neurons might be excited by inflammatory mediators, e.g. by bradykinin, NO (Michaelis et al., 1998; Ramer et al., 1998; Liu et al., 2000; Levy et al., 2000; Perkins & Tracey, 2000), and by cytokines (Cunha & Ferreira, 2003). The source of these inflammatory mediators might be white blood cells and Schwann cells around the damaged nerve fibres. The sympathetic nervous system does not activate primary afferents in normal tissue. Injured nerve fibres, however, might become sensitive to adrenergic mediators (Lee et al., 1999; Moon et al., 1999; Kingery et al., 2000). This cross-talk might occur at different sites. Adrenergic receptors might be expressed at the sensory nerve ending. A direct connection between afferent and efferent fibres (so-called ephapses) is considered. Sympathetic endings are expressed in increased numbers in the spinal ganglion after nerve injury. The cell bodies of the injured nerve fibres are surrounded by 'baskets', consisting of sympathetic fibres (Jänig et al., 1996). Currently, the best treatment is the application of drugs that reduce the excitability of neurons (e.g. carbamazepine or gabapentin)." The role of exercise in this regard needs to be further researched.*

### **2.1.2.3 Dysfunctional Pain**

According to Meyer (2007), large groups of chronic pain patients tend to demonstrate no existing peripheral abnormality or neurological deficit. The mechanism of pain has been described as an abnormal sensory processing of non-painful stimuli once the central nervous system has undergone changes that cause it to become sensitised (Bennett, 1999; Nielson & Nielson, 2003; Woolf, 2004). These disorders include the much described idiopathic pain disorders such as irritable bowel syndrome, chronic headaches, post-whiplash disorders, and fibromyalgia syndrome and others (Diatchenko *et al.*, 2006).

It is important to note that both neuropathic and dysfunctional pain may be present in the absence of an ongoing peripheral stimulus or 'organic cause'. It is wrong to assume that these patients are only psychologically unbalanced or even hysterical

(Nielson & Nielson, 2003; Woolf, 2004; Diatchenko *et al.*, 2006). It is important to note that both neuropathic and dysfunctional pain may be present in the absence of an ongoing peripheral stimulus or organic cause. It is wrong to assume that these patients are only psychological unbalanced or even hysterical OR both neuropathic and dysfunctional pain may be present in the absence of an ongoing peripheral stimulus or “organic cause”, and it is wrong to assume that these patients are only “psychological” or “hysterical” (Nielson & Nielson, 2003; Woolf, 2004; Diatchenko *et al.*, 2006).

#### **2.1.2.4 Mixed Pain**

These people include those displaying conditions such as cancer pain and low back pain, in particular low back pain following surgery, or failed back surgery syndrome where neuropathic, nociceptive and myofascial components may contribute to the patient’s increased pain perception (Shipton, 1999; Woolf, 2004; Meyer, 2007).

#### **2.1.3 Chronic Pain and its Effects**

Chronicity can be described in terms of persisting symptoms, disability and work status changes (Pincus *et al.*, 2002). Evidence of distorted local brain chemistry and functional reorganisation in chronic back pain patients support the idea that chronic pain could be understood not only as an altered functional state, but also as a consequence of central plasticity (Flor, 2003). ‘Plasticity’ is a term used to refer to changes that occur in the established nervous system. It has been observed that plasticity at several levels of the nervous system is related to the transmission of pain signals long after the original cause is gone, depriving pain of its functional role and thus becoming the disease itself (May, 2008).

Recent neurobiological findings suggest cortical reorganisation on a functional level (Grusser *et al.*, 2004). For example, amputation of a limb is often accompanied by phantom pain. In these patients the deafferentation leads to cortical reorganisation where the representational fields of adjacent areas move into the representation zone of the deafferented limb (Flor *et al.*, 2006). This ‘functional reorganisation’ has not only been detected in patients suffering from phantom limb pain but has also been observed in those suffering from chronic low back pain (May, 2008).



The incidence and prevalence of chronic pain do not appear to be diminishing, but may in fact be on the increase (Harstall & Ospina, 2003). Disability related to back pain has increased dramatically over the past 20 years due to influences in the form of psychological and social factors that influence adaptation to back pain early in the process (Waddell, 1996).

Pain and loss of function associated with musculoskeletal conditions primarily lead to disability (Woolf & Pfleger, 2003). For example, fear of pain and other psychosocial variables during an acute episode of back pain is related to chronic pain status at follow-up (Gatchel *et al.*, 1995; Klenerman *et al.*, 1995; MacFarlane *et al.*, 1999).

Disability is a very important factor regarding low back pain, for it has been reported that an improvement in the patient's subjective perception of functional disability is the most powerful predictor of treatment outcome (Pfungsten *et al.*, 1997).

Chronic pain has always been classified and diagnosed when it has lasted for more than six months (Schaible & Richter, 2004). However, an attempt has been made to define chronic pain by its characteristics rather than its duration (Schaible & Richter, 2004). Meyer (2007: 20) provides an accepted definition of chronic pain as state by the IASP: “...*pain that persists for longer than the time expected for healing, or pain associated with progressive, non-malignant disease, usually taken to be three months*”.

Others have defined chronic pain syndromes as any set of behaviours that involve the complaint of enduring or recurring pain which has persisted for longer than typical for an associated condition, is associated with an intermittent or chronic disease process, has inadequately responded to appropriate medical or invasive care, and is associated with significant and reliable impairment of functional status (Sanders *et al.*, 2005). Others have defined chronic pain by other sets of criteria. Sanders *et al.* (2005) reported chronic pain syndromes as any set of behaviours that involve the complaint of enduring or recurring pain which has persisted for longer than typical for an associated condition, is associated with an intermittent or chronic



disease process, has inadequately responded to appropriate medical or invasive care, and is associated with significant and reliable impairment of functional status.

This has given support to the hypothesis that chronic low back pain should not be regarded as an isolated spinal disorder (Kikuchi, 2008). It has been reported that two-thirds of subjects with chronic low back pain have at least another chronic pain conditions. About one-third has a diagnosable mental disorder (Von Korff *et al.*, 2005).

Patients who suffer from chronic pain syndromes may also demonstrate significant mood disturbances and/or anger-hostility, but these are not considered as necessary to make a diagnosis (Sanders *et al.*, 2005). Petersen *et al.* (2002) reported that there is a need for a classification system highlighted by Borkan & Cherkin (1996) to increase researchers' ability to identify differences in treatments for low back pain patients.

Petersen *et al.* (2002) reported that in diverse samples of non-specific low back pain patients, subgroups of patients for whom a specific treatment has been of benefit may be masked by subgroups for whom no measurable benefits was achieved. It can therefore be argued that the aims of such a classification system would be to identify patient characteristics that could predict the effects of different types of treatment procedures, and to distinguish clinically relevant subgroups for testing the relevance of specific treatment effectiveness.

Chronic pain can often persist long after the tissue trauma that triggered its onset, has healed and may be present in the absence of recognized ongoing tissue damage (Holdcroft & Jagger, 2005). Chronic pain is a dysfunctional response which mostly does not warn the individual of underlying disease or injury that will trigger an aversion response, and has thus accordingly been widely acknowledged as a disease in its own right (Niv & Devor, 2007; Kikuchi, 2008).

Chronic pain may be associated with an underlying chronic disease such as arthritis, but the largest group of chronic pain patients in the current epidemic in developing countries consists of the chronic pain syndromes of unknown etiology (Shipton,

1999). These pain syndromes have no confirmed laboratory evidence and are diagnosed on the basis of clinical criteria, such as the headache syndromes, irritable bowel syndrome, fibromyalgia and non-specific low back pain (Meyer, 2007). According to Meyer (2007) chronic pain may be associated with an underlying chronic disease such as arthritis. However, the largest group of chronic pain patients in the current epidemic in developed countries, comprises the chronic pain syndromes of unknown etiology (Shipton, 1999). These pain syndromes have no confirmed laboratory evidence and are diagnosed on the basis of clinical criteria, e.g. the headache syndromes, irritable bowel syndrome, fibromyalgia and non-specific low back pain.

Schaible & Richter (2004: 239) stated that “...*chronic pain might also result from a chronic disease and might then actually result from persistent nociceptive processes. It may be accompanied by neuroendocrine dysregulation, fatigue, dysphoria, and impaired physical and even mental performance (Chapman & Gavrin, 1999).* It has also been suggested that physical and sexual abuse in childhood can highly influence the incidence of chronic pain, especially in adulthood (McMahon *et al.*, 1997).

It is important to note that conservative treatment should be the first choice recommended treatment when treating patients with chronic low back pain (Zachrisson-Forssell, 1981; Hall & Iceton, 1983; Mayer *et al.*, 1985).

### **2.1.3.1 Psychological/Psychosocial Consequence of Chronic Low Back Pain**

There is increasing acceptance that psychosocial factors play a crucial role in the transition from an acute episode of low back pain to a chronic back disorder, as well as containing etiologic factors (Bigos *et al.*, 1994; Kendall, 1999). Schaible & Richter (2004: 238) stated that: “*In many chronic pain states the causal relationship between nociception and pain is not tight and the pain does not reflect tissue damage. Rather, psychological and social factors seem to determine the pain, e.g. in many cases of low back pain (Kendall, 1999).* It has been reported that back pain can be triggered by psychological problems such as distress (depression), poor health or excessive fear of illness (Carragee *et al.*, 2000). However, the tendency to consider chronic

pain as either psychological or physical is a false implication, because both play a role in most chronic pain disorders, although the balance between organic pathology and psychosocial contributions may differ in different pain disorders (Meyer, 2007). According to Meyer (2007) however, the tendency to consider chronic pain as either psychological or physical implies a false dichotomy - both play a role in most chronic pain disorders, although the balance between organic pathology and psychosocial contributions may differ in different pain disorders.

The emotional component of pain is complex and is influenced by past experiences, patient beliefs and fears (Turk, 2002). Catastrophising, described as an exaggerated orientation towards pain stimuli and pain experience (Sullivan *et al.*, 1995), is considered to be a maladaptive coping mechanism. It is interesting that it has been described as an explanatory concept for variations in pain and depression in chronic pain patients (Keefe *et al.*, 1989).

According to Meyer (2007) negative beliefs and an attitude of hopelessness may generate maladaptive illness behaviour with increased pain reporting (Baumann, 1994). Improvement in the understanding of the psychosocial aspect of suffering in chronic pain has led to an improvement in the effectiveness of rehabilitation programmes (Kääpä *et al.*, 2006).

Fear avoidance behaviour also plays an important part in the development of chronic low back pain. Fear avoidance beliefs are suggested to contribute to the development of chronic low back pain earlier than previously believed (Klenerman *et al.*, 1995; Linton *et al.*, 2000; Fritz *et al.*, 2001; Picavet *et al.*, 2002; Sieben *et al.*, 2002). It is reported that patients who perceive pain in a threatening, catastrophic manner (as in signs of tissue damage) are much more likely to encounter pain related fear and anxiety. This will consequently lead to escape or avoidance behaviours to situations that they perceive to be potentially harmful (Thomas & France, 2007). Confrontation and avoidance have been suggested as the two extreme responses to the fear of pain (Lethem *et al.*, 1983). Consequences of avoidance include escape and avoidance behaviour, resulting in poor performance and muscle reactivity, physical disuse and guarded movements (Vlaeyen & Linton, 2000).

Distress has been identified as a predictor of chronic pain and disability. This effect is independent of clinical factors, such as pain and function at baseline level measurements (Dionne *et al.*, 1995; Cherkin *et al.*, 1996; Dionne *et al.*, 1997; Epping *et al.*, 1998; Thomas *et al.*, 1999; Linton *et al.*, 2000; Pincus *et al.*, 2002). Distress deals with aspects such as anxiety, somatisation and depression in general, whereas fear avoidance beliefs focuses more on specific back pain related anxiety (Grotle *et al.*, 2006). Distress contributes about the same amount as fear avoidance beliefs to the changes in variance in disability scores (Grotle *et al.*, 2004).

It has also been reported that distress is a significant prognostic factor of non-recovery at three months post-onset, whereas fear avoidance beliefs are not (Grotle *et al.*, 2005). Thus, it has been reported that leaving out the distress variable may result in an over-optimistic conclusion regarding the prognostic role of fear avoidance beliefs (Grotle *et al.*, 2006).

Thomas and France (2007) has reported that over the long-term, the avoidance of activities of everyday life that are perceived by the person to increase pain or a risk for re-injury is repeatedly reinforced, can cause anxiety symptoms towards these activities. It can then be argued that pain-related fear and anxiety eventually contribute to symptoms of disuse and disability.

Grotle *et al.* (2006) reported that fear avoidance beliefs for physical activity follow the same clinical pattern as pain and pain-related disability. Consistent with this model, studies have shown that individuals suffering from chronic low back pain who also experience high levels of pain-related fear demonstrates sub-maximal performance on a variety of physical tasks (Waddell *et al.*, 1993; Klenerman *et al.*, 1995; Vlaeyen *et al.*, 1995; Crombez *et al.*, 1999; Geisser *et al.*, 2000; Burns *et al.*, 2000; Sieben *et al.*, 2002; Fritz & George, 2002; Swinkels-Meewisse *et al.*, 2003; Al-Obaidi *et al.*, 2000; Al-Obaidi *et al.*, 2003).

Moore *et al.* (2000) have reported that approximately two months after consultation with a primary care physician for back pain, approximately 64% of patients still feel that incorrect movement could lead to a serious problem, and over 46% still feel they

could suffer disability for a long time. This type of thinking can contribute to the avoidance of activities or to additional health care visits, making the levels of anxiety of a patient an important target of clinical care. These patients however feel that they have a reason for concern, as it has been shown that guarded movements and hyperactivity in the lumbar paraspinal muscles are directly correlated with pain-related fear (Maffey-Ward *et al.*, 1996; Main & Watson, 1999), as they feel discomfort with movement.

Thomas and France (2007) reported that prior studies have usually defined physical impairment on the basis of reduced exertion. This classification may show that individuals with pain-related fear alter the manner in which they move in an effort to avoid pain or further damage (Thomas & France, 2007). As an example, research of motor coordination have shown that individuals with low back pain have reduced peak velocity and acceleration of the upper body (Marras *et al.*, 1995b; Ferguson *et al.*, 2000; Tawfik, 2001), and limitations and asymmetries in range of motion (Bishop *et al.*, 1997; Magnusson *et al.*, 1998).

These kinds of changes in motor behaviour may possibly reflect adaptations that reduce spinal load, and to avoid large forces on healing muscles and irritated joints. More likely they are a learned response due to fear avoidance behaviour with no actual reduction in spinal loading (Thomas & France, 2007).

Whichever the case, such adaptations may contribute to increased risk for subsequent injury. Thomas and France (2007) reported that this may explain the association between pain-related fear and disability. The authors also suggested that patients with high pain-related fear tend to adopt alternative movement patterns and strategies to avoid motion of the lumbar spine when performing everyday reaching movement tasks. Also, patients performing movement tasks have no preference to move at slow or fast speeds, but it is a specific position that they aim to avoid (Thomas & France, 2007).

Simmonds and Dreisinger (2003) reported that an approach to exercise will be influenced by the individuals beliefs about their back pain. Beliefs differ from person to person, as some people will ignore their own pain and discomfort and carry on

with normal every day activities while others will stop all forms of daily activity and seek medical attention. If certain activities aggravate pain, certain types of people will avoid activities or even attempt to avoid those activities that they expect will cause pain. These beliefs will influence the probability of these people to partake in exercise treatment modalities, and it can be severely compromised if their beliefs are negative.

Psychological issues of motivation and fatigue must be taken into consideration in relation to isometric extension endurance testing before a statement can be made regarding their diagnostic value (Moreau *et al.*, 2001). It has been suggested that a disadvantage of endurance testing is its dependence on subject motivation which can be influenced by a subjects perception of being able to maintain a given sub-maximal target force as measured to their own perceived limit of endurance (Biering-Sorensen, 1984; Mannion & Dolan, 1994; Ng & Richardson, 1996; Van Dieen & Heijblom, 1996).

Many factors influence a person's perception of exertion. Moreau *et al.* (2001) reported that some subjects who possess personalities motivated by achievement and competition tend to be better at endurance based tests, traits of which include competitiveness (Hellandsig, 1998), self-motivation (Raglin *et al.*, 1990), leadership qualities (Clingham & Hilliard, 1987), ambition, organisation, deference, dominance, endurance, self-control, tough-mindedness (Ogilvie, 1968), lower rates of perceived exertion (Beaudoin *et al.*, 1998), less negative feelings during endurance tasks (Beaudoin *et al.*, 1998), the ability to activate an emotion appropriate for the task (Forbes, 1986), and control of fatigue and pain (Forbes, 1986). These traits might mean a critical difference when measuring poor endurance performers from good performers, even when all other physiological variables are similar (Moreau *et al.*, 2001).

Moreau *et al.* (2001) further reported that the effect of personality traits on endurance variables may extend beyond athletes to non-athletes. This could imply that certain personality types are susceptible to working beyond their endurance and recuperative capacities (Moreau *et al.*, 2001). These people include business executives, doctors, lawyers, accountants, clergymen and housewives (Rhoads,

1977). Personality traits that are often seen in those who perform well on endurance tasks may suggest that white-collar workers perform better because they are more psychologically suited to the tasks and they respond as a result (Moreau *et al.*, 2001).

To add to the psychological problem, most patients presenting with first-time low back pain will be diagnosed according to a scientific standpoint as having non-specific low back pain (simple back pain) (Staal *et al.*, 2005). Many of these patients, however, may desire a more somatic name for their pain, such as a 'slipped disc' in order to justify their complaints and to show to others that their pain is indeed real and that they can prove it (Staal *et al.*, 2005).

The assumed disadvantages of receiving a somatic diagnostic label or name is that besides the possible lack of validity, they may enhance pain-related fears such as fear avoidance beliefs and encourage a dependant 'sick-role' (Staal *et al.*, 2005). The message that needs to be communicated to the patients is that their pain is real but that it does not necessarily imply harm. This should not prevent patients from being active (Frank *et al.*, 1998).

### **2.1.3.2 Physical Changes and Deconditioning**

Leboeuf-Yde (2004) reported that a sedentary lifestyle is probably one of the most causative factors for low back pain, as lack of physical activity can lead to reduced muscle strength and flexibility, as well as having an undesirable effect on proprioception. All of these factors can contribute to a maladapted and weakened spine. Such persons are therefore more prone to injuries (Leboeuf-Yde, 2004).

Empirical research has demonstrated that physiological changes such as muscle dysfunction occur in the lumbar spine in conjunction with an initial episode of pain. These changes remain after the pain episode has subsided (Hides *et al.*, 1996; Hodges & Richardson, 1996). People with low back pain often have declining muscle strength as well as endurance, along with greater atrophy of the back muscles. This may compromise the functional capacity of the spine and increase the likelihood of re-injury (Jackson & Brown, 1983; Reid *et al.*, 1991; Parkkola *et al.*, 1993b). This includes stiffness of the lumbar spine-pelvic-femoral unit, decreased muscle strength



and endurance, a loss of adequate cardiorespiratory response to increased physical exertion as well as a inhibition of neuromuscular activation (Mayer *et al.*, 1985).

Thomas and France (2007) proposed that changes that occurs due to low back pain includes the restriction of low back motion, which leads to changes in extensibility of noncontractile connective tissues, muscle recruitment patterns, or alteration in potential feedback signals provided from the muscle spindles and mechanoreceptors in the paraspinal muscles (Thomas & France, 2007). Structural changes in any of these variables could increase the risk of exacerbation of symptoms by interfering with the ability of the individual to control spinal motion (Thomas & France, 2007).

Limitation of the range of motion at a joint may possibly result in reduction in length of peri-articular connective tissues as well as changes in the surrounding muscles (Lieber, 2002). Kendall *et al.* (1993) reported that at least in theory, this may lead to a condition called 'adaptive shortening', which results in tightening that occurs because of the muscle remaining in a shortened position. Unless the opposing antagonistic muscle is able to pull the part back to the neutral position or some type of outside force is exerted to increase the length of the shortened muscle (e.g. stretching), it will remain in a shortened position (Kendall *et al.*, 1993), and can lead to a decrease in muscle length and a corresponding limitation in range of motion (Kendall *et al.*, 1993).

Thomas and France suggested that individuals who demonstrate high levels of pain-related fear who continue to restrict motion may be at greater risk for pain and re-injury when faced with physical challenges that necessitate them to move the spine into and beyond its restricted range of motion. The authors has thus proposed that there are increased demands on shortened connected tissues and muscles that are no longer able to adequately maintain the integrity of the motion segment through normal range of motion, especially in movement tasks that require high velocities (Thomas & France, 2007).

In the past, wasting and denervation of the multifidus muscle in acute and sub-acute low back pain populations has been suggested (Hides *et al.*, 1994; Hides *et al.*, 1996). For example, the multifidus muscle denervation and atrophy have been



reported in patients with lumbar disc herniation injuries (Mattila *et al.*, 1986; Rantanen *et al.*, 1993). This may have an effect on muscle receptors, which in turn will influence trunk proprioception and position sense (Leinonen *et al.*, 2003).

Previous studies have found that a denervation of the multifidus muscle is frequently present in patients suffering from sciatica as well (Mattila *et al.*, 1986; Rantanen *et al.*, 1993). This denervation often occurs in the lower limb muscles innervated by the specific nerve root. The subsequent reinnervation process may take longer than three months (Leinonen *et al.*, 2003).

There is a large body of evidence suggests that poor back extensor muscle endurance and co-ordination cause excessive mechanical loading on the passive structures of the lumbar spine and can exacerbate existing low back pain symptoms (Wilder *et al.*, 1996; McGill, 1997; Taimela *et al.*, 1999). It has been shown previously that lumbar fatigue restricts the ability to sense a change in lumbar rotation location (Kankaanpää *et al.*, 2005). Dynamic testing has also shown pathologically low muscle activities and asymmetries in dynamic movements in back pain patients (Hoyt *et al.*, 1981; Cram & Steger, 1983).

Interestingly, patients with low back pain have shown unsuccessful the ability to increase their paraspinal activity level during the Valsalva manoeuvre or a sit-up test (Soderberg & Barr, 1983). This feature, however, was found in subjects and healthy controls, but subjects with low back pain demonstrates poorer ability to sense a change in lumbar position than healthy controls even when they are not fatigued (Kankaanpää *et al.*, 2005).

Sung *et al.* (2008) reports that subjects with low back pain demonstrate a greater level of fatigue of the erector spinae muscles at the thoracic part than at the lumbar part regardless of gender. The thoracic portion of the erector spinae muscle may play a significant role in spinal endurance for subjects with low back pain compared to subjects without low back pain (Sung *et al.*, 2008). Thus, the thoracic part of the erector spinae muscle shows higher fatigue levels than the lumbar portion. This is in part due to the lumbar region extending over a longer period during tests such as the Sorensen back extensor endurance test (Sung *et al.*, 2008). There seems to be a

phase after a fatiguing task during which the existing information on lumbar position and its changes is faulty (Taimela *et al.*, 1999; Leinonen *et al.*, 2003).

Adequate blood supply is obviously the most essential for the lumbar muscles to withstand fatigue and prevent the loss of sense organ functions that are driving the co-coordinative feedback mechanisms. The most essential factor for the lumbar muscles to withstand fatigue and prevent the loss of sense organ functions that are driving the co-coordinative feedback mechanisms is adequate blood supply (Kankaanpää *et al.*, 2005). Suitable rehabilitation strategies based on lumbar erector spinae muscle fatigability may advance the retraining of trunk musculature and prevent re-injury in patients who have a history of low back pain (Sung *et al.*, 2008).

Chronic low back pain has been linked with histomorphological and structural changes in the paraspinal muscles, to an extent that the back muscles are smaller, contain higher levels of fat, demonstrates a degree of selective muscle fibre atrophy (Verbunt *et al.*, 2003) and their blood circulation may be constrained because of calcific deposits in the abdominal aorta and vertebral arteries (Kauppila *et al.*, 1997; Kauppila *et al.*, 2004). Consequently, the lumbar paraspinal muscles are weaker (Hakkinen *et al.*, 2003) and show signs of excessive fatigability, as compared to those without low back pain (Mannion *et al.*, 1997; Greenough *et al.*, 1998; Humphrey *et al.*, 2005).

Also, reduced co-ordination of paraspinal muscles has been associated with chronic low back pain and with excessive lumbar muscle fatigability (Wilder *et al.*, 1996; Taimela *et al.*, 1999; Leinonen *et al.*, 2003). These changes are generally considered to be a consequence of disuse and deconditioning that are secondary to pain and illness, a process that has been named the 'deconditioning syndrome' (Nachemson & Lindh, 1969; Thorstensson & Arvidson, 1982).

Recent studies suggest that pain induced muscle spasms and reflex inhibition of the paraspinal muscles may also lead to the physical deconditioning (Hides *et al.*, 1996; Indahl *et al.*, 1997; Verbunt *et al.*, 2003). Radebold *et al.* (2000) has suggested that patients with low back pain have increased reaction times in muscle response patterns to sudden trunk loading when compared to healthy controls (Radebold *et*

*al.*, 2000). Patients seem to be able to maintain agonistic muscle contraction while their antagonistic muscles become concurrently activated. There seems to be no definite change from agonistic to antagonistic status during movement (Radebold *et al.*, 2000).

Delayed reaction times to unexpected and sudden loading (as in slipping or tripping) for subjects with low back pain could be interpreted either as a predisposing factor to injury or as a consequence of soft tissue damage that necessitates an altered motor control approach to stabilise the lumbar spine (Magnusson *et al.*, 1996; Wilder *et al.*, 1996). Previous soft tissue injuries may have irreversibly damaged proprioceptors and therefore an adequate fast reflex response to a sudden load may not be possible (Radebold *et al.*, 2000). Proprioceptors that have been irreversibly damaged by past soft tissue injuries has an inadequate reflex response time to sudden loads, and thus protective response is not possible.

Accordingly, a response time delay must be compensated for by an altered recruitment pattern. Co-activation of the antagonistic and agonistic muscle groups has been reported to stiffen and consequently stabilise the lumbar spine in order to protect the spine from injury (Bergmark, 1989a; Gardner-Morse *et al.*, 1995; Cholewicki & McGill, 1996; Gardner-Morse & Stokes, 1998; Quint *et al.*, 1998). The continued contraction of the agonistic muscles, which are similar in nature to muscle spasms, in subjects with low back pain serve to increase the joint stability and consequently provide an adequate method of compensation to protect them from pain and harm progression due to lumbar spine instability (Radebold *et al.*, 2000).

Oldervoll *et al.* (2001) reported that poor muscle strength in the thigh, dorsal and abdominal muscles can cause back pain indirectly, in that weakness-related fatigue in the thigh muscle at the end of a working day can cause a person to lift an object with straight knees and a bent back rather than bending at the knee and hip joint (Oldervoll *et al.*, 2001). This style of lifting can cause amplified load on the passive structures in the low back (Nutter, 1988).

Kendall *et al.* (1993) reported that painful conditions such as the so-called ‘periformis syndrome’ can indicate how nerve irritation associated with muscle stiffness can cause pain, because the applied pressure can be through pressure on nerve roots, nerve trunks, nerve branches or nerve endings and can be caused by some adjacent firm structure such as bone, cartilage, fascia, scar tissue or stiffened muscles applying pressure to the nerves. All of this can lead to pain.

Apkarian *et al.* (2004) has reported that it seems that a reduction in grey matter has been observed in the dorsal-lateral prefrontal cortices (DLPFC) of the brain. This phenomenon has been observed bilaterally, as well as in the right thalamus. Neural degeneration has been identified as a possible causative factor rather than outright tissue shrinkage.

Schmidt-Wilcke *et al.* (2006) reported an increase in gray matter in the thalamus with an additional level of decrease in the dorsal-lateral pons and the somatosensory cortex. Pain intensity and the unpleasant experience of pain has been suggested to be involved with the reduction in grey matter in the brainstem, rather than the timeframe of the pain experience.

Chronic pain has been identified as a multi-factorial condition that can lead to chronic disability the longer the pain persists. Psychological, social and occupation factors all become involved the longer that pain persists, and they can all contribute to a longer term problem (Waddell *et al.*, 1996).

### **2.1.3.3 The Concept of Central Sensitisation**

This concept is very important in the management of chronic pain syndromes, for it is a key mechanism involved in the persistence of pain.

#### **2.1.3.3.1 The Role of Acute Pain in Central Sensitisation**

Meyer (2007: 22) stated that: “*Acute pain is a normal biological response to injury or tissue trauma and a signal of ongoing or impending tissue damage, e.g. post-operatively. It protects the organism from further injury and promotes healing after injury*”. The cause of acute pain has to be treated appropriately due to the fact that it is a symptom of something else (Shipton, 1999; Woolf, 2004). Meyer (2007)

reported that unnecessary suffering and increased morbidity will result if acute pain is left untreated. It can also cause an increase in recovery time. Meyer (2007: 22) further stated that: *“There is increasing recognition that long-term changes may occur within the peripheral and central nervous system following the noxious input of painful stimuli”*. Meyer (2007:22) wrote: *“There is increasing recognition that long-term changes may occur within the peripheral and central nervous system following the noxious input of painful stimuli. Even brief interval of untreated acute pain can induce long-term neuronal remodelling and central sensitisation (“plasticity”), and may lead to chronic pain in some patients”*. This sensitization of the nervous system then changes the response of the body to further sensory input which then causes it to become more sensitive to pain impulses and even harmless stimuli can trigger pain perception (Melzack & Wall, 1965; Shipton, 1999; Carr & Goudas, 1999; Woolf, 2004).

Verdu *et al.* (2008: 2613) stated that: *“In inflammatory pain, the peripheral terminals of nociceptors are subjected to major changes in their chemical environment leading to peripheral sensitization. The numerous inflammatory mediators (prostaglandins, cytokines, bradykinin, amines, and neurotrophic factors, etc.) can directly sensitize the terminal in a way that it becomes more receptive.”*

#### **2.1.3.3.2 The Physiology of Central Sensitisation**

Meyer (2007:22) reported that *“Central sensitisation is a complex process involving many neurochemical and molecular processes”*. It seems to be triggered by intense activation of nociceptors as well as by humoral factors released by inflamed peripheral tissue (Woolf, 1983). Schaible & Richter (2004: 240) stated that: *“Pathological nociceptive input often causes central sensitisation. This is an increase of excitability of spinal cord neurons (Woolf, 1983).”*

Verdu *et al.* (2008:2612) stated that: *“In chronic pain syndromes, the activation of multiple pathophysiological mechanisms leads to a shift towards hyperexcitability of the somatosensory system.”* Schaible & Richter (2004: 240) further stated that: *“Hyperexcitable spinal cord neurons are more susceptible to peripheral inputs and respond, therefore, more strongly to stimulation. Central sensitisation amplifies the processing of nociceptive input and is thus an important mechanism that is involved*

*in clinically relevant pain states. It consists of the following phenomena: a) increase of the response to input from the injured or inflamed region; b) increase of the responses to input from regions adjacent to and even remote from the injured/inflamed region, although these areas are not injured/inflamed; c) expansion of the receptive fields of the spinal cord, i.e. the total area from which the neuron is activated, is enlarged. Presumably the latter accounts for secondary hyperalgesia, i.e. hyperalgesia in normal tissue surrounding the injured/inflamed area (Schaible et al., 1987)."*

According to Meyer (2007) central sensitization is a complex process involving many neurochemicals and molecular processes, and is induced by the release of neuropeptides such as substance P and glutamate, which then activates the NMDA-receptor complex (Bennett, 1999; Bonica, 1953; Carr & Goudas, 1999). The subsequent intra-cellular events may lead to long-term neuronal changes, characterized by a more sensitive nervous system and hyperalgesia.

Schaible and Richter (2004:240-241) reported that: "*Pressure on this area causes a response of the neuron. Stimulation of the surrounding adjacent area does not cause a response, although some afferent fibres from this fringe area project to the same neuron. Under normal conditions, synaptic activation by these afferents is too weak to evoke a suprathreshold response. During injury, nociceptors in the receptive field are sensitised, and their increased activity causes activation and sensitisation of the spinal cord neuron. When the spinal cord neuron is rendered hyperexcitable, the weak inputs from the adjacent regions outside the original receptive field are sufficient to excite the spinal cord neuron, and, hence, the receptive field shows an expansion. Another consequence of peripheral inflammation and spinal sensitisation is that, in the spinal segments with input from the lesioned/injured regions, a higher proportion of neurons respond to stimulation of peripheral tissue (Woolf, 1983; Schaible et al., 1987; Neugebauer & Schaible, 1990; Grubb et al., 1993)."*

Normally, when the nociceptive input is removed, central sensitization decreases as well, but it has been reported that central sensitization may last longer than the peripheral nociceptive stimulation (Sandkühler & Liu, 1998). Hours and days after injury the pain signal can remain sustained by transcriptional changes in the dorsal



horn. These changes, restricted to the activated synapse or spread to adjacent synapses, are responsible for pain produced by low-threshold afferent inputs and pain in regions beyond the tissue injury (Verdu *et al.*, 2008). It has been suggested that as long as a painful nociceptive signal remains that central sensitization will remain until the signal is removed (Sandkühler & Liu, 1998). According to Verdu *et al.* (2008) however, the pain signal can still remain by transcriptional changes in the dorsal horn, and these changes, restricted to the activated synapse or spread to adjacent synapses, are responsible for pain produced by low-threshold afferent inputs (allodynia) and pain in regions beyond the tissue injury (secondary hyperalgesia), and even when the nociceptive signal has been removed.

It is theorised that nociceptive inputs may trigger a so-called long-term potentiation and a persistent increase of synaptic efficacy will occur. Such a process could account for pain states that persist even when the peripheral nociceptive process has been removed (Sandkühler & Liu, 1998). Repetitive end-range loading of pain-sensitised spinal tissue may act towards the maintenance of a chronic pain disorder in the absence of pathoanatomic abnormalities (O'Sullivan *et al.*, 2003). After nerve injury, the amount of input produced by peripheral ectopic activity, in addition to changes in gene expression, will contribute to central sensitisation (Verdu *et al.*, 2008). It has been proposed that a process of persistent increased synaptic efficacy can occur which can trigger a long-term potentiation. This type of response could account for painful situations that occur when the peripheral nociceptive signal has been removed (Sandkühler & Liu, 1998). O'Sullivan *et al.* (2003:1077) stated that “...repetitive end-range loading of pain-sensitised spinal tissue may act towards the maintenance of a chronic pain disorder in the absence of pathoanatomic abnormalities”. According to Verdu *et al.* (2008) after nerve injury, the amount of input produced by peripheral ectopic activity, in addition to changes in gene expression, will contribute to central sensitization.

## **2.2 The Problem of Low Back Pain**

Low back pain is a very common disorder and is one of the most common types of musculoskeletal pain that affects a larger number of people from many walks of life (Frymoyer & Cats-Baril, 1991; Van Tulder *et al.*, 2000).

Punnett *et al.* (2005:2) stated that “...*low back pain has been defined as any non-traumatic musculoskeletal disorder affecting the low back. It includes all back pain, regardless of the diagnosis, that was not secondary to another disease or injury cause (e.g. cancer or motor vehicle accident). It included lumbar disc problems (displacement, rupture) and sciatica*”.

However, it has been reported that the definition of low back pain may vary substantially across studies and prevalence estimates may therefore vary substantially (Loney & Stratford, 1999). Such differences in definitions are not likely to affect the estimation of relative risk as long as they are applied in a regular manner within each study (Punnett *et al.*, 2005).

Low back pain can result from various sources, which can include either one serious event such as major trauma or even multiple episodes of microtrauma. It can include a muscular and joint component and can involve single or multiple sites and could continue from anything up to a couple of weeks, months or even span a lifetime (Simmonds & Dreisinger, 2003). A specific diagnosis is thus very difficult, resulting in signs and symptoms being the main considerations that will determine a course of treatment, because even the most objective data is vulnerable to subjective interpretation of its' significance (Kendall *et al.*, 1993).

There exists a lack of understanding with regard to the multifactorial nature and specific cause of low back problems, and this is shown in the large variety of obtainable treatments, ranging from medically oriented invasive treatments such as injection therapy and surgery to more psychological approaches such as behaviour based approaches (Nelemans *et al.*, 2001; Van Tulder *et al.*, 2001).

Radebold *et al.* (2000:947) reported that: “*Many of the factors associated with low back pain are mechanical. These factors either cause low back problems initially or aggravate them by increasing the risk of recurrence and are thus important for disability considerations*”. Burton (2005) has suggested that low back pain tends to present itself as a disorderly collection of periods with increased discomfort due to symptom elevation which is interchanged with periods of less symptom activity



(Croft *et al.*, 1998; De Vet *et al.*, 2002; Hestbaek *et al.*, 2003a). For a group of people, these symptoms can become chronic, as will the disability associated with it.

### 2.2.1 Lifetime Occurrence of Low Back Pain

Kankaanpää *et al.* (2005) reported that because of the lack of understanding with regards to the underlying mechanisms of low back pain, even modern scanning techniques such as X-rays or MRI scanning procedures along with clinical examinations cannot make a specific diagnosis due to a lack of sufficient information. Burton (2005) reported that in 85% of people that pathology and/or neurological encroachment cannot be attributed directly to pain. The tendency then is to make either a diagnosis that is descriptive of symptoms rather than pathology or a diagnosis of nonspecific low back pain (Nachemson, 1992; Deyo, 1994).

The prevalence for low back pain in industrial developed countries over a lifetime, which will account for at least one episode over a lifetime is reported to be at around 84-85% (Walker, 2000; Simmonds & Dreisinger, 2003). Hildebrandt *et al.* (2004) reported that after an initial episode of low back pain 44-78% of patients will suffer from future relapses in pain and 26-37% will suffer relapses in occupational absence. Sweden reports a prevalence of 69% (Ihlebaek *et al.*, 2006). In South Africa prevalence has been reported to be at 63.9% (Van Vuuren *et al.*, 2005).

Hildebrandt *et al.* (2004) reported that around 11-12% of patients that suffer from low back pain are actually disabled by their pain, and similarly to other, also report that specific causes are unusual, with less than 15% of all cases of low back pain that can be directly related to a specific diagnosis. But, more important from the aspect of the current study, about 5-10% of those who do suffer an initial episode of low back pain will go on to become chronic and thus long-term (Linton *et al.*, 2005).

Goldby *et al.* (2006) reported that over a one year time period, patients who suffer a first-time episode of low back pain that 20% will be without symptoms and 70-80% of patients have at least one reoccurring episode of low back pain (Klenerman *et al.*, 1995; Croft *et al.*, 1997). Many of these patients will continue to have recurring episodes of low back pain that has been reported up to 20 years (Carey *et al.*, 2000). About 3-4% of these patients will develop a specific chronic pain syndrome, but the

largest percentage is those with chronic low back pain, and they compose an estimated 73-77% of all the reported low back pain problems.

Punnett *et al.* (2005) reported that around 37% of all low back pain episodes worldwide is attributable to occupational risk factors. Epidemiologic evidence suggests that patients with chronic low back pain disorders have recurrences from which they rapidly recover over a period of time (Goldby *et al.*, 2006).

### **2.2.2 Impact of Low Back Pain**

Reported estimates have shown that disability resulting from low back pain can contribute to a significant economic burden due to direct and indirect costs that can exceed \$25-50 billion per annum in the United States (Frymoyer & Cats-Baril, 1991; Frymoyer, 1992) with associated productivity losses accounting for about \$28 billion per annum (Rizzo *et al.*, 1998). Even personal costs are high, as it is estimated that people who suffer from low back pain incur health care costs that are around 60% higher than those without low back pain (Luo *et al.*, 2004).

In the United Kingdom, primary health care consultation for low back pain is the second highest leading cause of health care consultation (Deyo & Phillips, 1996). It also appears that the economic, societal and public health effects of low back pain appear to be increasing (Louw *et al.*, 2007). Punnett *et al.* (2005) reported that low back pain is not responsible for premature mortality but it can lead to severe consequences, such as substantial levels of disability. This is of particular concern if these consequences are suffered at a young age.

Low back pain incurs billions of dollars in medical expenditure each year (Childs *et al.*, 2004). This economic burden is of particular concern for poorer nations such as those in Africa where HIV/AIDS takes preference in terms of funding towards health care management and funds are thus restricted to deal with musculoskeletal problems (Walker, 2000). It has been suggested that most of the research on low back pain has been conducted in the developed world that doesn't have the same social and economic conditions such as those in Africa and other developing nations (Worku, 2000). Louw *et al.* (2007) suggested that Africa is considered to be a developing continent, and it is characterized by factors such as racial, economic and

social heterogeneity. It can thus be argued that reported differences in the prevalence of low back pain between developed and developing nations can be obscured by differences in social and economic structure as well as genetic diversity.

Lopez *et al.* (2006) reported that that prevalence among Africans are also influenced by other factors that are unique to Africa, such as the high HIV/AIDS incidence, types of working tasks unique to Africans as well as poor nutrition. Even in South Africa the cost and impact are high. Sick days taken because of back pain costs companies around R1.2 billion a year, and sick days taken is second only to flu with around 6.4% of all sick days taken (SAPA, 2009).

Significant levels of low back pain results in considerable levels of associated disability, which places restrictions on usual activities of daily living. This includes the ability of the person to continue to work (Katz, 2006). Western societies are influenced greatly by worker absenteeism and disablement, which results in the largest amount of related economic costs (Andersson, 1999). Burton (2005) reported that the greatest number of back pain episodes usually return to work in due time (Phelps *et al.* 2004), but recurrent and chronic low back pain are considered to be responsible for a large portion of the total number of work absenteeism. Work absenteeism accounts for a large proportion of the socio-economic burden of low back pain.

Staal *et al.* (2005:492) stated that "*Return to work (in the case of work absenteeism due to low back pain) should not only be considered as an important treatment goal and outcome measure in research, but also an important part of the treatment. While working, the disabled worker realises that he/she is still able to be active, despite discomfort. Being at work, in a partial or full capacity, also draws the attention of the disabled worker away from negative issues such as pain, and helps to decrease the focus on disablement*".

Burton (2005) reported that from half the days lost from work due to back pain that it is for a short period of time and they return to work in less than 7 days. This accounts for 85% of people who are absent from work due to low back pain. The other 15% accounts for the other half of days missed and the workers are off from

work for longer than a month. This has important economic implications, in that patients who are absent from work for more than six months has a 50% chance of returning to work. This number becomes lower the longer the person is away from work. Being off from work for more than one year has a 25% chance of returning to work and being off for longer than two years has less than a 5% chance of returning to work (Bergquist-Ullman & Larsson, 1977).

The amount of available academic literature on the epidemiology of low back pain is increasing, but it reported that most of the studies are done in developed, high-income countries, and much less is thus known about the rest of the world (Volinn, 1997). Prevalence in countries such as Australia and the United States for low back pain ranges anywhere from 26.4%-79.2% (Deyo *et al.*, 2006). It is assumed that the prevalence in a developing continent such as Africa is much lower, but this could be due to a lack of reporting (Omokhodion & Sanya, 2003; Omokhodion, 2004; Gilgil *et al.*, 2005).

Walker (2000) reported that a systematic review into the low back pain prevalence globally identified 56 studies, of which only 8% were conducted in developing countries, and only one study was done specifically in Africa. It is clear that there is a lack of research done in developing nations, and this presents a significant shortcoming (Walker, 2000; Gilgil *et al.*, 2005). This lack of hard data presents a problem, as it is predicted that the greatest increase in low back pain incidence in the next decade will occur in developing nations (Louw *et al.*, 2007). Regardless, Louw *et al.* (2007) reported that even due to the lack of evidence, there appears to be not much difference between Africa and developed nations with regards to incidence of prevalence.

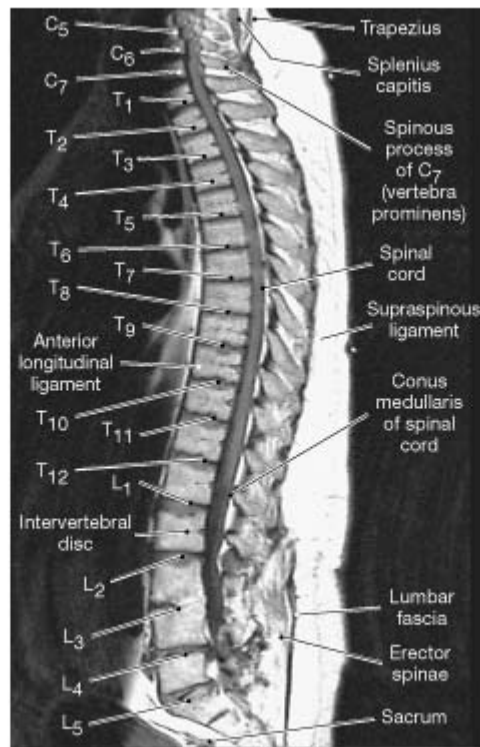
## **2.3 Low Back Anatomy**

Understanding the anatomy and function of the lower back is crucial to understanding dysfunctional conditions.

### **2.3.1 The Bone Structure**

The lumbar spine contains a total of five vertebrae (McGill, 2002). The construction of a vertebra consists of relatively stiff cortical bone on the outside walls and more

deformable cartilage plates that are approximately 0.66 mm thick on the sides of the vertebrae (Roberts *et al.*, 1989). These sides of the vertebrae are known as endplates, and they are porous in terms of their construction in order for nutrients such as oxygen and glucose to be transported. The inside of the barrel of the vertebral body is filled with cancellous bone (Roberts *et al.*, 1989).

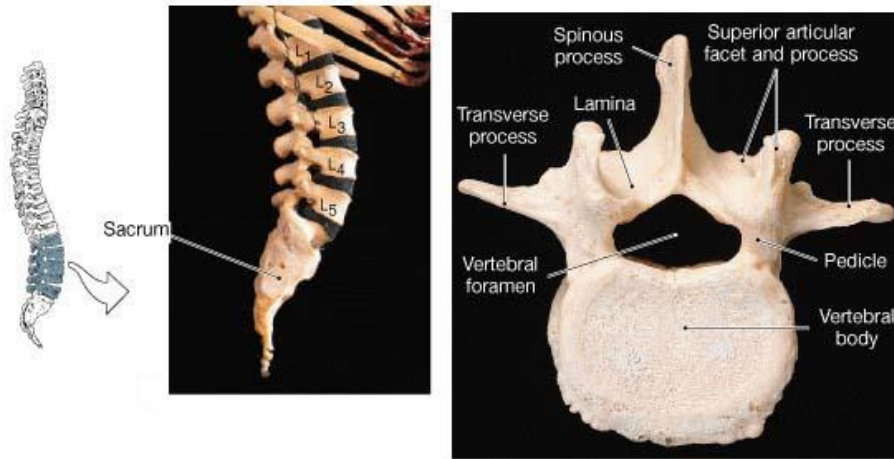


**Figure 2.2:** A Radiological View of the Spine (Martini, 1998)

Within the cancellous bone of the vertebral body, the trabecular arrangement is aligned with stress trajectories that develop during activity, and three orientations domination, with one being vertical and two are oblique (Gallois & Japoit, 1925).

McGill (2002) reported that the ability of the vertebral body to cope with compressive loads or either to fail under extreme loads is determined by its construction. The vertebral loads will remain fairly ridged under compressive loads, but the pressure will affect the nucleus of the disc (Nachemson, 1960). This will cause the cartilaginous endplates of the vertebra to bulge inward, and to consequently compress the cancellous bone (Brinckmann *et al.*, 1989). This cancellous bone tends to fail first (Gunning *et al.*, 2001). This is then considered to be a failure

determination point of the spine, at least when the spine is not located at the end range of motion. It is unlikely that the annulus itself will be injured by this mechanism.



**Figure 2.3:** The Lumbar Vertebrae (Martini, 1998)

Upon axial compression, as the endplates bulge into the vertebral bodies, these columns experience compression and appear to bend. Under excessive compressive load the bending columns will buckle as the smaller bony transverse trabeculae fracture (Fyhrie & Schaffler, 1994). In this way the cancellous bone may rebound back to its original shape or to at least 95% of the original unloaded shape when the load is removed, even after suffering fracture and delamination of the transverse trabeculae (Fyhrie & Schaffler, 1994). This architecture appears to afford excellent elastic deformation, even after marked damage, and then to regain its original structure and function as it heals (McGill, 2002). McGill (2002: 47) stated that: “...upon axial compression, as the endplates bulge into the vertebral bodies, these columns experience compression and appear to bend”. Under greater than normal loads, the smaller bony transverse trabeculae will fracture under greater than normal spinal loads, allowing the cancellous bone the opportunity to return to at least 95% of its’ original pre-loaded shape, even if fractures and delamination occurs (Fyhrie & Schaffler, 1994). This organization tends to offer good elastic deformation



properties, in that the original structure and function can return as healing starts to occur after injury (McGill, 2002).

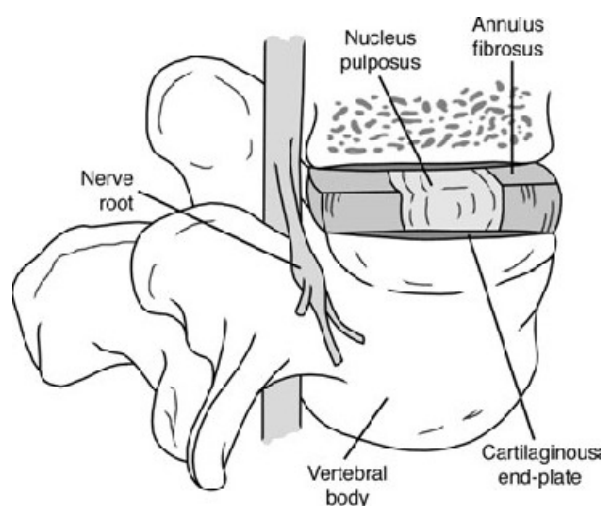
McGill (2002: 51) reported that: "*The posterior elements of the vertebrae (pedicles, laminae, spinous processes and facet joints) have a shell of cortical bone but contain a cancellous bony core in the thick sections. The transverse processes project laterally together with a superior and an inferior pair of facet joints. On the lateral surface of the bone that forms the superior facets are the accessory and mamillary processes that, together with the transverse processes, are major attachment sites of the longissimus and iliocostalis extensor muscle groups. The facet joints are typical synovial joints, in that the articulating surfaces are covered with hyaline cartilage, and are contained within a capsule*". Around the rim of the facet, fibro-adipose enlargements are found at the proximal and distal edges, and these structures have been implicated as structures that could lock a facet joint (Bogduk & Engel, 1984).

McGill (2002:48) also reported that: "*Micro-fracturing of the trabeculae can occur with repetitive loading at levels well below the failure level from a single cycle of load*." Lu *et al.* (2001) demonstrated that cyclic loading at 10% of ultimate failure load caused no damage or change in stiffness, but with 20 000 cycles of load at 20-30% of the ultimate failure load, both stiffness and energy absorbed at failure were decreased. Highly repetitive loads, even at quite low magnitudes, appear to cause micro-damage. According to Lu *et al.* (2001) cyclic loading at 10% of failure load seems to cause no damage or change in stiffness. It was found however, that at 20 000 cycles of load at 20-30% of ultimate failure load, there was a marked decrease in both stiffness and energy absorbed at failure. For this it would appear that even at low levels, highly repetitive loads appear to cause micro damage.

### **2.3.2 The Intervertebral Disc**

The intervertebral discs are cartilaginous, articulating structures between the vertebral bodies and link them together, allowing movement (flexion, extension and rotation) in the otherwise rigid anterior portion of the vertebral column (Roberts *et al.*, 2006; Prithvi Raj, 2008). The intervertebral discs are the links between vertebrae that allow for movement such as flexion, extension and rotation. The vertebral column

would have been a rigid structure if not for the intervertebral discs. They are cartilaginous in terms of their structure (Roberts *et al.*, 2006; Prithvi Raj, 2008). The discs are approximately 7-10 mm thick and 4 cm in diameter in the anterior-posterior plane in the lumbar region of the spine (Twomey & Taylor, 1987; Roberts *et al.*, 1989). McGill (2002:53) reported that: “*The intervertebral disc has three major components: the nucleus pulposus, annulus fibrosus and the endplates*”. The discs consist of a thick an outer ring called the annulus fibrosus which consists of a thick outer ring of fibrous cartilage. This surrounds an inner core known as the nucleus pulposus, which is more gelatinous (Roberts *et al.*, 2006; Prithvi Raj, 2008). The discs consist of a network of collagen fibres that are composed of mostly Type 1 and Type 2 fibres, and is responsible for about 70% of the dry weight of the annulus and about 20% of the nucleus weights. This arrangement provides tensile strength to the discs and connects the tissue to the bone (Eyre & Muir, 1977).



**Figure 2.4** : The Intervertebral Disc Between Two Adjacent Vertebrae  
(Prithvi Raj, 2008)

### 2.3.2.1 The Nucleus Pulposus

McGill (2002:53) reported that “...*the nucleus has a gel-like character with collagen fibrils suspended in a base of water and various mucopolysaccharides giving it both viscosity and some elastic responses.*” The nucleus pulposus is located in the central part of the disc and contains randomly organized collagen fibres (Inoue, 1981) as well as radially organized elastin fibres, all of which is organized in highly hydrated aggrecan-containing gel (Yu *et al.*, 2002).

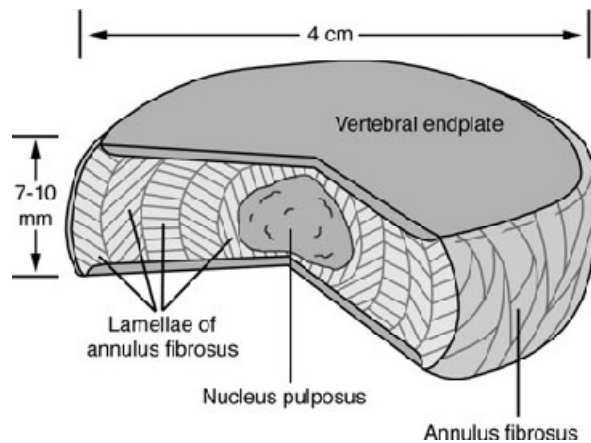


McGill (2002:53) reported that: “*While there is no distinct border between the nucleus and the annulus, the lamellae of the annulus become more distinct, moving radially outward. The collagen fibres of each lamina are obliquely orientated (the obliquity runs in the opposite direction in each concentric lamella). The ends of the collagen fibres anchor into the vertebral body with Sharpey’s fibres in the outermost lamellae while the inner fibres attach to the end plate*”. To add to this, Roberts *et al.* (2006:10) reported that: “*Collagen fibres continue from the annulus into the adjacent tissues, tying this fibrocartilaginous structure to the vertebral bodies at its rim, to the longitudinal ligaments anteriorly and posteriorly, and to the hyaline cartilage end plates superiorly and inferiorly. The cartilage end plates in turn lock into the osseous vertebral endplates via the calcified cartilage with few, if any, collagen fibres crossing the boundary*”.

### **2.3.2.2 Annulus Fibrosis**

The construction of the annulus fibrosis consists of around 15-25 concentric rings (or Lamellae) (Marchand & Ahmed, 1990) with the collagen fibres being organized in a parallel fashion with each lamella (Prithvi Raj, 2008). The fibre orientation is around 60 degrees to the vertical axis which alternates left and right of the axis in each adjacent lamellae (Prithvi Raj, 2008).

Elastin fibres are located between the lamellae. These fibres are suggested to assist the disc in returning to its’ original position after either flexion and/or extension. Because the elastin fibres run radially from one lamella to the next, they also bind the lamella together (Yu *et al.*, 2002). The cells of the outer region of the annulus are aligned parallel to the collagen fibres, are elongated and thin and tend to be fibroblast-like, but tend to be more oval towards to inner portion of the annulus (Prithvi Raj, 2008).



**Figure 2.5:** The Structure of an Intervertebral Disc (Prithvi Raj, 2008)

### 2.3.2.3 The Endplate

This morphologically distinct region consists of a thin layer, usually 1 mm thick, consisting of hyaline cartilage which interfaces the disc and the intervertebral body (Prithvi Raj, 2008). The collagen fibres within it run horizontal and parallel to the vertebral bodies, with the fibres continuing into the disc (Roberts *et al.*, 1989).

### 2.3.2.4 Properties and Function of the Intervertebral Discs

McGill (2002) reported that the intervertebral disc acts as a whole to allow for about six degrees of motion between the vertebra by exhibiting hydrostatic structure properties. The annulus and the nucleus work together when the spine is subjected to bending and compression to supporting compressive forces by pressurizing the nucleus and thus applying a hydraulic force to the end plates vertically and laterally to the annulus. This will cause an outward bulging by the annulus collagen fibres and causes them to tense.

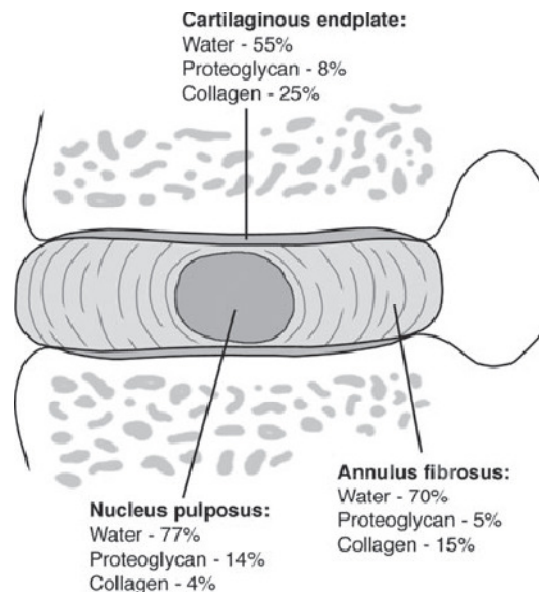
Roberts *et al.* (2006) reported that normal intervertebral discs contain an extracellular matrix that is responsible for about 1% of the total volume of the disc. This matrix is interspersed by a small number of cells, which are believed to consist of at least two phenotypical distinct populations (Chelberg *et al.*, 1995).

Roberts *et al.* (2006:11) reported that: "...the cells are morphologically different. Those in the annulus fibrosis and cartilage endplates are more elongated and fibroblast-like compared to those of the nucleus pulposus, which are more rounded

*or oval and chondrocyte-like, sometimes with a capsule around them.*” The cells seem to be simple in appearance, but might be much more complex in function, with long thin cell processes which could be able in sensing mechanical strain (Errington *et al.*, 1998).

Roberts *et al.* (2006: 11) also reports that: “...*in addition, these two populations behave differently, such as in their response to applied loads or in synthesizing different matrix molecules when grown in culture.*” Nucleus pulposus cells are commonly produced only type-2 collagen in alginate beads, while annulus fibrosus cells produce both type -1 and type-2 (Chelberg *et al.*, 1995).

The normal, healthy adult disc does contain a limited number of nerves and blood vessels, but these are limited only to the outer few millimetres of the annulus fibrosus. There are also a number of mechanoreceptors present, which demonstrates a similar morphology of Golgi tendon organs, some Ruffini receptors and an ever smaller amount of pacinian corpuscles (Roberts *et al.*, 1995).



**Figure 2.6:** The Composition of the Intervertebral Disc Structures  
(Prithvi Raj, 2008)

The main function attributed to the intervertebral disc is a mechanical function, in that the loads from body weight and muscle activity are transmitted through the vertebral column (Prithvi Raj, 2008).

### **2.3.3 Movements of the Vertebral Column**

Kendall *et al.* (1993:23) reported that: “*Vertebral articulations include the bilateral synovial joints of the vertebral arches where the inferior facets of one vertebra articulate with the superior facets of the adjacent vertebra, and the fibrous joints between successive vertebral bodies united by intervertebral fibrocartilaginous discs. Movement between two adjacent vertebrae is slight and determined by the slope of the articular facets and the flexibility of the intervertebral discs. The range of motion of the column as a whole, however, is considerable and the movements permitted are flexion, extension, lateral flexion and rotation.*”

Motion of the vertebral column is provided by the intervertebral discs, which includes flexion (forward and backwards) as well as rotation (Prithvi Raj, 2008). The ability of the vertebral column to move differs in the thoracic and the lumbar region. In the lumbar region, the range of motion for greater flexion, extension and lateral bending ability about the three axis of motion, while the thoracic region demonstrates greater ability to rotate (McGill, 2002).

### **2.3.4 The Model of Spinal Stability and Instability**

Stability can be defined as “...*the ability to maintain intervertebral and global torso equilibrium despite the presence of small mechanical disturbances and/or small neuromuscular control errors*” (Granata & England, 2006:E271).

Instability of the lumbar spine in those with chronic low back pain has been identified as a significant causative factor for pain (Friberg, 1987). This type of lumbar instability can be thought of as a specific region of laxity around the neutral resting position of the spinal neutral zone (Panjabi, 1992). This also includes an inability of the stabilizing mechanism as a whole to maintain the neutral zone of the vertebral column within physiological limits, which means that there is no deformity of any structures, any neurological deficit or debilitating pain (Panjabi, 1992).

The stability of the spine can be compromised by a decrease in motion segmental stiffness by as little as 10% (Gardener-Morse *et al.*, 1995). This stability is provided by osteoligamentous structures and muscles since the spinal structure has to cope with large amounts of pressure due to complex loading patterns in all three dimensions simultaneously, and if unprotected, the vertebral column is susceptible to injury (Arokoski *et al.*, 2004). The ability of the vertebral column to generate stability and be decreased by injury and chronic mechanical derangement (Oxland & Panjabi, 1992).

Trunk muscles must compensate during normal motion by changing their typical activation pattern to maintain stability (Panjabi, 1992). To achieve this, the muscles of the lumbar and abdominal regions must have to precisely control motion by generating enough muscle tension and correct timing to optimizing loading on the spine and to avoid overload injury (Bergmark, 1989a; Crisco & Panjabi, 1991; Callaghan & McGill, 1995; Gardner-Morse *et al.*, 1995; Kaigle *et al.*, 1995; Cholewicki & McGill, 1996; McGill, 1997; Gardner-Morse & Stokes, 1998).

Hasegawa *et al.* (2008) reported that the concept of lumbar stability is difficult to define objectively. Radiological evaluation remains unclear and controversial, since radiological evaluations are performed regularly on degenerative lumbar spines and clear diagnoses are unclear and undefined (Knutsson, 1944; Morgan & King, 1957; Lindahl, 1966; Nachemson *et al.*, 1979; Dupuis *et al.*, 1985; Frymoyer & Selby, 1985; Dvorak *et al.*, 1991; Iguchi *et al.*, 2003).

Biplanar, cineradiographic and fluoroscopic measurements provide some additional information on disordered motion patterns (Stokes *et al.*, 1981; Pearcy *et al.*, 1985; Kanayama *et al.*, 1996; Okawa *et al.*, 1998; Harada *et al.*, 2000; Takayanagi *et al.*, 2001). These dynamic approaches cannot be used to draw a biomechanical conclusion about instability, because no information about the load deformation relationship can be determined from the images (Hasegawa *et al.*, 2008). Hasegawa *et al.* (2008) reported that measurement techniques such as biplanar, cineradiographic or fluoroscopic measurements provide some additional information on chaotic motion. Unfortunately, no biomechanical conclusion or diagnosis can be

made about instability because no information about the load deformation mechanisms can be concluded from these dynamic approaches.

Correct levels of muscle stiffness cannot alone verify biomechanical segmental properties of the spine, as other variables, including measurement of the neutral zone are needed to make accurate assessments (Panjabi, 1992). It has been suggested that up to about 20-30% of all reported chronic low back pain problems demonstrates verified lumbar segmental instability (O'Sullivan, 2000; Beith *et al.*, 2001). Spinal stability or instability can be an indicator of the ability of a person to activate and perform a co-contraction of the deep local muscle system (O'Sullivan, 2000; Beith *et al.*, 2001).

Granata and England, (2006: E271) states that research indicates that three subsystems contribute to spinal stability as initially proposed by Panjabi (1992):

- The passive contributions from the spinal ligaments, discs, and bone.
- The steady-state active muscle recruitment contribution to spinal stability.
- Neural feedback system that includes active and voluntary responses.

### **2.3.5 The Passive Stabilising Structures of the Spine: Bone, Ligaments and Fascia**

When the spine is in a position of neutral lordosis, e.g. neither being in flexion or extension, only muscles are responsible for mechanical support for the spine, but as the spine is flexed and rotates, the passive structures of the spine are also stressed and the forces on these tissues changes the injury mechanics (McGill, 2002). Spinal ligaments are an important spinal stabilizer, and they have been suggested as possessing an important proprioceptive role in spinal mechanics (Solomonow *et al.*, 2000).

In vitro, under compressive loads of about 90N, the osteoligamentous lumbar spine becomes mechanical unstable, and this load is less than the weight of the human upper body (Crisco *et al.*, 1992). Too much load and stress on the joints and ligaments of the spine will result if the muscle is inadequate (Gracovetsky *et al.*, 1985; Panjabi, 1992; Saal, 1992).

McGill (2002: 49) stated that: *"Both the disc and the vertebrae deform while supporting spinal loads. Under excessive compressive loading, the bulging of the endplates into the vertebral bodies also causes radial stresses in the endplate sufficient to cause fracture in a stellate pattern"*. When these fractures in the endplates are big enough, the liquid from the nucleus can move into the fracture spaces into the vertebral body (McGill, 1997). It can happen that a local area under the endplate can collapse to form the classic Scmorl's node injury, which has been described as an injury associated with compression of the spine when there is limited motion of the spine.

McGill (2002) reported that fascia of the lumbodorsal region has attachment sites on the posterior superior iliac spines as well as on the spinous processes, and some of these fascial connections cross the midline, which has been implicated in force transmission by completing a brace-like structure around the abdomen. This structure has been researched, and it has been found that it may function more as an extensor muscle retinaculum rather than being an active extensor due to the well-developed collagen fibre construction (Bogduk & Macintosh, 1984). Unstable behaviour and possible tissue injury is thus prevented by this mechanism due to muscles activation from the abdominal wall and the latissimus dorsi adding tension to the fascia and thus stiffness to the spine (McGill, 2002).

### **2.3.6 Dynamic Spine Stability: The Muscles Supporting the Spine**

The concept of different trunk muscles playing different roles in the provision of dynamic stability to the spine was proposed by Bergmark (1989a). It has hypothesised the presence of two muscle systems in the maintenance of spinal stability.

The global muscle system consists of muscles that provide trunk stability without being directly attached to it. These include the muscles of the rectus abdominis, external oblique and the thoracic portion of the lumbar iliocostalis, which are large torque-producing muscles which are not capable of having direct influence on the spine, but provide excellent spinal stability (Bergmark, 1989a).



The other important group of muscles is the so-called local muscle system, which is responsible for providing stability to the segmental portions of the spine and directly controls the lumbar segments. This is due to the fact that this group of muscles attaches directly to the lumbar vertebrae and includes the multifidus, transversus abdominis and the posterior fibres of the internal oblique (Bergmark, 1989a).

The local muscle group (transversus abdominis, internal oblique and the multifidus) have been reported to be tonically active during upright postures and during active spinal motion (Oddsson & Thorstensson, 1990; Cresswell *et al.*, 1992). The transversus abdominis is considered one of the most important trunk stabilizers, because it has been found that it seems to be tonically active regardless of the position of the trunk, loading of the spine or direction of movement (Cresswell *et al.*, 1992).

Leinonen *et al.* (2003) reported that in order to prevent low back injuries, it is important for correct muscle control and movement sensation to be of working order. The erector spinae group serves an important function, as they seem to have a greater mechanical advantage in some portions of the muscle than others due to multiple attachment sites, and this is important for maintaining an upright posture of the trunk (Bogduk, 1991; Bogduk *et al.*, 1992).

Because the erector spinae are attached to the lumbar vertebrae and directly extend the lumbar spine, they have been reported to be very important from an anatomical point of view (Sung *et al.*, 2008). This serves as an effective lever arm for performing back extension due to its attachment to the spinous processes of the lumbar vertebrae, and this portion of the erector spinae contributes about 20% of the lumbar spine extension moment at L4 and L5 (Bogduk *et al.*, 1992).

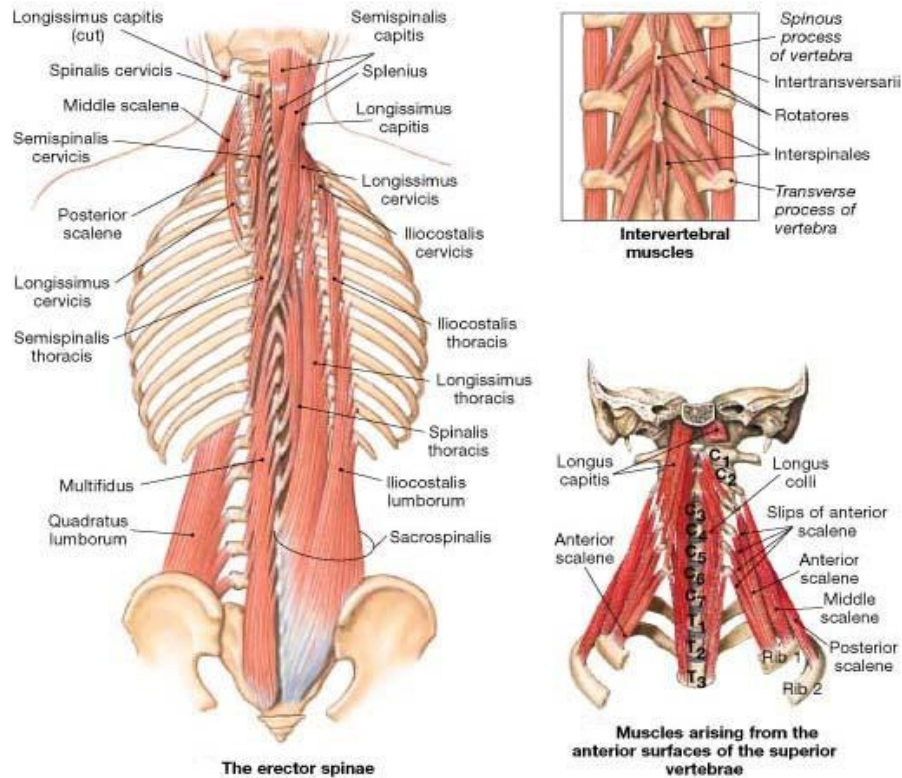
The erector spinae portion in the thoracic area consists of the thoracic components of the longissimus thoracis and iliocostalis lumborum and have been found to also generate moment arms across the L4-L5 joint due to the fact that they also cross the lumbar spine (McGill & Norman, 1986; Bogduk *et al.*, 1992). The muscles have a stabilizing effect by surrounding the injured joint segment and thus reduce sudden



kinematic behaviour, especially in the neutral region of the spine where muscles are under reduced tension (Kaigle *et al.*, 1995).

Intra-abdominal and abdominal spring force is created during spinal stability which is achieved with trunk flexor extensor muscle activation (Panjabi *et al.*, 1989). In patients with low back pain, the alteration in function of these muscles causes a change in function as compared to healthy controls without back pain (Hides *et al.*, 1996; Hodges & Richardson, 1996). This dysfunction presents itself clinically as an imbalance between the local and global muscle systems (Bergmark, 1989b). This then leads to a theoretical reduction of the deep local muscle systems' ability to maintain effective stability and control lumbar stability (Goldby *et al.*, 2006).

The lumbar extensor muscles that stabilize the spine of those with back pain have been found to be impaired in terms of their function and co-ordination (Magnusson *et al.*, 1996; Wilder *et al.*, 1996; Hodges & Richardson, 1999; Leinonen *et al.*, 2001). Function-wise there is also a reduction, as it has been found that those with low back pain demonstrates an increase in reaction time during and after sudden load transmission as compared to healthy controls (Magnusson *et al.*, 1996; Wilder *et al.*, 1996). Those with low back pain demonstrate hostomorphic and structural changes to the type-2 muscle fibres. These muscle fibres have been reported to show atrophy due to disuse and deconditioning (Parkkola *et al.*, 1993a; Ng *et al.*, 1998; Mannion, 1999; Mannion *et al.*, 2000).



**Figure 2.7:** The Muscles of the Spine (Martini, 1998)

Leinonen *et al.* (2003) reports that the proprioceptive information of body movements originates from the muscle spindles, Golgi tendon organs, joint receptors and cutaneous receptors (Rothwell, 1994; Schmidt & Lee, 1998). Upper (Hodges & Richardson, 1996; Hodges *et al.*, 1999; Hodges *et al.*, 2001) and lower (Hodges & Richardson, 1998) limb movements cause trunk muscle activation via a feed-forward mechanism. When certain trunk muscles activate, they prepare to potentially bear load and to maintain postural stability.

The muscles responsible for this movement include the transversus abdominis muscle and the transversospinal muscles. It has been found that these muscles seem to activate shortly after perturbation. More significantly, it has been suggested by others that they seem to activate shortly before the muscles that are responsible for gross limb movement (Belen'kii *et al.*, 1967; Cordo & Nashner, 1982; Friedli *et al.*, 1988; Zattara & Bouisset, 1988; Aruin & Latash, 1995; Hodges & Richardson, 1996; Hodges *et al.*, 1999; Hodges *et al.*, 2001).

It has been proposed that possible faults, even only small faults in the proprioception and position sense ability can lead to tissue overload and injury. Insufficient activation can lead to abnormal loading across the surfaces of joints and can lead to early degenerative disease (Gross, 1987; Cholewicki & McGill, 1992; Forwell & Carnahan, 1996).

Movement of the limbs causes reactive forces that are imposed on the spine which are equal in the magnitude but opposite in the direction of those forces that are responsible for the movement (Belen'kii *et al.*, 1967; Cordo & Nashner, 1982; Bouisset & Zattara, 1987; Crisco & Panjabi, 1991). It has been proposed that the body anticipates limb movement and implements direction specific strategies to control reactive forces and to prepare the body for perturbation (Aruin & Latash, 1995; Moseley *et al.*, 2002). It has also been found that the transversus abdominis isn't influenced by the direction of any reactive moments, and is considered to be responsible in contributing to spine stiffness (Hodges & Richardson, 1997).

Due to the importance of the activity of the transversus abdominis, Richardson *et al.* (2002) has reported that the concept has become the basis of the specific exercise treatment techniques (Richardson *et al.*, 1999). The ability to co-contract the transversus abdominis and the lumbar multifidus independently of the other larger trunk muscles has become the goal of many exercise regiments. This exercise is based on evidence of the stability roles of the different implicated muscles (Goel *et al.*, 1993; Kaigle *et al.*, 1995) as well as on evidence that the transversus abdominis seems to function independently of the other abdominal muscles (Hodges & Richardson, 1997). A very low level of muscular activation is actually performed during this action (Richardson *et al.*, 1999). Progression of treatment has consisted, in principle, of increasing the patient's ability at performing this independent deep muscle contractions action while minimising the contribution of the other trunk muscles.

Reasons have been proposed for the motivation to limit the activation of lumbar stability exclusively to the transversus abdominis in the form of lumbar paraspinal electromyography which has been used to estimate extensor force generation and

spinal muscle compression forces during lifting activities (Örtengren *et al.*, 1981; Dolan & Adams, 1993). It has been found that high levels of muscle activity could lead to unfavourable compressive forces acting on the spine, which in theory can cause possible injury (Callaghan *et al.*, 1998; McGill, 1998).

It has been reported that contraction of the muscles of the low back needs only to be about 25% of the maximal voluntary contraction (MVC) to provide optimal stiffness (Cresswell *et al.*, 1994). Others have reported that to improve muscle performance, contractions needs only to be around 30-40% of the MVC (Richardson *et al.*, 1999). Thus, the therapeutic exercises selected for a rehabilitation programme must be carefully selected as to not increase the risk of injury on the low back but be sufficient to increase the performance of the muscles.

### **2.3.6.1 Muscles Involved in Spinal Stability**

Production of high force at fast speeds as well as the provision of stability and postural control is indicative of the functional capacity of the muscles of the low back, and these functions are dependent on anatomical, physiological, biomechanical and neural variables (Thorstensson & Carlson, 1987; Lieber, 1992; Enoka, 1994).

Muscle fibre composition, cross-sectional area, fibre length, pennation angle and muscle mass are some of the anatomical and physiological variables that are considered important. Important biomechanical variables are moment arm through which the muscle acts, velocity of the muscle contraction as well as the type. Compartmentalisation and recruitment strategies are considered neural parameters (Lieber, 1992; Enoka, 1994).

Ng *et al.* (1998) reported that anatomical, physiological, biomechanical and neural variables in combination results in the diverse functions among individual muscles. But another factor, namely muscle fibre composition is one of the most important elements that is indicative of the functional capacity of a specific muscle (Johnson *et al.*, 1973; Thorstensson & Carlson, 1987; Rome *et al.*, 1988).

The lumbar region consists of the iliocostalis lumborum, longissimus lumborum and the multifidus (from lateral to medial), which forms the paraspinal muscle group of

the lumbar region (Hyun *et al.*, 2007). To control posture and to stabilize joints has been identified as the primary function of these muscles (Bajek *et al.*, 2000).

Richardson *et al.* (2002: 399) reported that: “*The local muscle system includes deep muscles such as the transversus abdominis and the lumbar multifidus that are attached to the lumbar vertebrae and sacrum and are capable of directly controlling the lumbar segments*”.

The next layer of muscles has been called the global muscle system. This group of muscles consists of muscles such as the external oblique and the erector spinae, which are bigger muscles and are located more towards to surface of the body and are more responsible for producing and controlling trunk movements (Bergmark, 1989a). The trunk basically consists of back extensor muscles that extend the trunk, lateral flexors that bend it sideways and the anterior abdominal group that flexes the trunk. All of these muscles have a trunk stabilizing function, but the extensor group has been recognized as being the most important (Kendall *et al.*, 1993).

Richardson *et al.* (2002) reported that conventional exercises are generally designed to increase the strength of the global muscle system. A more specific exercise approach is thus recommended to target the local muscle system in order to improve the dynamic stability function in order to provide muscle stiffness to support the spinal segments during functional postures and activities.

Different muscles exert types of force production on the trunk. In the upward direction, the erector spinae and the quadratus lumborum are primarily responsible for force generation. In the upward and anterior direction, the rectus abdominis and external oblique are responsible. The gluteus maximus and the hamstring muscle group generates force in a downward posterior direction, while the hip flexors (which includes the tensor fasciae latae, rectus femoris and sartorius) pulls downward and anteriorly (Kendall *et al.*, 1993).

Kendall *et al.* (1993:70) stated that: “*The low back muscles act with the hip flexors (especially the psoas with its direct pull from the lumbar spine to the femur) to tilt the pelvis down and forward (anterior tilt). They are opposed in action by the combined*

*pull of the anterior abdominals pulling up anteriorly, and the hamstrings and gluteus maximus pulling down posteriorly to level the pelvis from a position of anterior tilt. Hip abductors on one side and lateral trunk muscles on the other side combine in action to tilt the pelvis laterally: right abductors (gluteus minimus and medius) pull downward on the right side of the pelvis as left lateral trunk muscles pull upward on the left side, and vice versa. These actions are assisted by hip adductors on the same side as the lateral trunk muscles.”*

It has been previously reported that the transversus abdominis, which is very important in contributing to spinal stability, shows strong abnormal motor control abilities in those with low back pain (Hodges & Richardson, 1996; Hodges & Richardson, 1998). Changes in multifidus composition has, which could affect the deep portion of the muscle, has also been observed (Rantanen *et al.*, 1993; Hides *et al.*, 1996; Moseley *et al.*, 2002).

The deep fibres of the multifidus and the transversus abdominis are controlled very similarly in healthy subjects without low back pain. Both of these muscles can contribute to the control of intervertebral motion, but the body needs all of the trunk muscles to stabilize the spine. Since both the deep multifidus and the transversus abdominis seem not to be specific to any direction of movement or movement from limbs, they are considered to be the most important spinal stabilizers (Richardson *et al.*, 1999; Moseley *et al.*, 2002).

Training of the transversus abdominis and multifidus muscles is believed to be an important component in the rehabilitation of patients with low back pain (Hides *et al.*, 1996; Hodges & Richardson, 1996; O’Sullivan *et al.*, 1997; O’Sullivan *et al.*, 1998; Hides *et al.*, 2001; Richardson *et al.*, 2002). The simultaneous contraction of the deep abdominal muscles and the lumbar multifidus can enhance the segmental stability of the spine during functional tasks and maintaining neutral spine postures by providing a type of dynamic corset for the lumbar spine, enhancing thus its stability irrespective of the position of the spine (Aspden, 1992).

The skeletal muscles of the body have been divided into two subcategories. Type 1 fibres have been described as being slow-twitch fibres. Type 2a and Type 2b are



described as being fast-twitch fibres. These distinctly different muscle fibres also show different reactions to mechanical loading and will be described in detail in the next section (Brooke & Kaiser, 1970; Billeter *et al.*, 1980; Herbison *et al.*, 1982; Yoshihara *et al.*, 2001).

Ng *et al.* (1998: 390) reported that: “*Type 1 fibres are slow twitch fibres that possess properties of high oxidative, low glycolytic capacity and are relatively resistant to fatigue. Type 2b fibres are most commonly known as fast twitch fibres with the characteristics of low oxidative, high glycolytic capacity and are prone to fatigue. Type 2a fibres possess features in between 1 and 2b fibres.*” Generally, type 2 fibres are larger in cross-sectional area (Enoka, 1994) and produce greater force (Rothstein, 1982; Jones *et al.*, 1989) than type 1 fibres. It has also been reported by others that type 2 fibres are on average larger than type 1 fibres in the limb muscles (Polger *et al.*, 1973). However, it has been shown that type 1 fibres in the lumbar multifidus muscle are much larger than the type 2 fibres found in the same muscle (Bagnall *et al.*, 1984; Mattila *et al.*, 1986; Yoshihara *et al.*, 2001).

Ng *et al.* (1998) reported that Type 1 muscle fibres predominate in tonic muscles and tend to assume a postural stability, sustained contraction and endurance activities. In contrast, Type 2 muscle fibres have fast twitch properties and are involved in high speed, forceful movements of short duration (Johnson *et al.*, 1973; McCafferty & Horvath, 1977; Herbison *et al.*, 1982). They occupy a relatively larger area in muscles responsible for fast movements (Johnson *et al.*, 1973) and appear to dominate in non-postural muscles such as the triceps brachii (Johnson *et al.*, 1973).

Paravertebral muscles function to control posture and to stabilize joints, which are the responsibility of the type 1 slow twitch fibres as they predominate in these muscles. They are suited to this kind of task since they are fatigue resistant (Fidler *et al.*, 1975; Thorstensson & Carlson, 1987). These muscles have been shown to be active in almost every kind of normal activity performed daily, but they are rarely ever fully contracted, regardless of modern lifestyle requirements (Bajek *et al.*, 2000).

Ng *et al.* (1998) reported that in general, 54-73% type 1 fibres are found in longissimus, iliocostalis and multifidus muscles of healthy subjects. Those with low

back problems tend to show changes in percentages. Longissimus is between 54-68%, 47-68% in multifidus and 68-69% in erector spinae muscles respectively.

A greater percentage of type 1 muscle fibres in the back muscles could possibly be explained as a postural control function. This type of activity will require endurance capacity to maintain, especially in an upright position against gravity which has to be maintained (Ng *et al.*, 1998). Due to a functional difference between the lumbar and thoracic portions of the muscles in postural control, a higher percentage of type 1 fibres have been identified in the thoracic portion of the muscles (Širca & Kostevc, 1985).

Ng *et al.* (1998) wrote that due to the S-shape of the spinal column, the line of gravity is more anterior to the rotational axis of the intervertebral joints in the thoracic vertebrae than in the lumbar spinal area. This implies that much larger flexion moments are generated at the thoracic levels (Joseph & McColl, 1961).

An investigation of the relevant muscles involved is of clinical importance, because it has been suggested that there is a functional differentiation between individual back muscles in the production of torque and the provision of stability (Thorstensson & Carlson, 1987; Panjabi *et al.*, 1989).

#### **2.3.6.1.1 Multifidus**

The multifidus muscle has been identified as being primarily involved in providing the required stiffness for the lumbar spine, and is consequently regarded as the most important of the back extensor muscles (Wilke *et al.*, 1995). Observations made with electromyographic measurements have suggested that the multifidus extends the length of the spine (Floyd & Silver, 1951; Joseph & McColl, 1961; Morris *et al.*, 1962; Donisch & Basmajian, 1972).

According to Kendall *et al.* (1993):

Multifidi

*“Origin: In the sacral region, posterior surface of the sacrum, medial surface of posterior superior iliac spine and the posterior sacroiliac ligaments as well as through the transverse processes of the L5 through to the C4 vertebrae.*



***Insertion:*** *The muscle spans two to four vertebrae and inserts into the spinous processes of an above vertebrae.”*

Moseley *et al.* (2002) reported that the greatest amount of activity in the multifidus muscle has been shown by surface EMG measurements to be during rotation and extension of the vertebral column or during resistance of lumbar flexion (Floyd & Silver, 1951; Joseph & McColl, 1961; Arokoski *et al.*, 1999). Direction specific activity during standing trunk movements and limb movements in prone subjects have been demonstrated in intramuscular EMG studies (Morris *et al.*, 1962; Pauly, 1966). It has to be noted that it is however, impractical to use surface EMG measurements to measure the deep fibres of the multifidus (Moseley *et al.*, 2002).

The lumbar multifidus has been identified as a major contributor to stabilization and lumbar spine control (Moseley *et al.*, 2002). Various types of studies, such as biomechanical models and in vitro studies have identified a function of maintaining spinal stiffness and intervertebral motion control in all planes of motion, especially the sagittal and frontal planes of motion (Panjabi *et al.*, 1989; Crisco & Panjabi, 1991; Wilke *et al.*, 1995; Moseley *et al.*, 2002).

The multifidus has been suggested to contribute to rotation of the spine as well as maintaining postural integrity (Morris *et al.*, 1962; Donisch & Basmajian, 1972; Dofferhof & Vink, 1985; Kalimo *et al.*, 1989). The maintaining of postural control and in the end segmental integrity seems to be dependent on the ability of the muscles to respond during changes in structural integrity, has been shown by studies in vivo from porcine models that spinal motion has to be controlled by the multifidus after induced instability by ligamentous disruption (Kaigle *et al.*, 1995).

Moseley *et al.* (2002) reported that the multifidus has five fascicles that arise from the spinous process and lamina of each lumbar vertebra and descends in a caudolateral direction (Macintosh *et al.*, 1986). The most superficial fibres of each fascicle cross up to five segments and attach caudally to the ilia and sacrum. The deep fibres attach from the inferior border of a lamina and from the inferior edge of the spinous process (Macintosh *et al.*, 1986; Bogduk, 1997). They cross a minimum of two segments to insert into a mamillary process and facet joint capsule (Macintosh

*et al.*, 1986; Bogduk, 1997). These are the deepest muscle fibres in the lumbar spine because there are no rotation muscles in this deep region (Bogduk, 1997). Biomechanically, the superficial fibres are more distant from the centres of lumbar vertebra rotation and have an effective moment arm for extension of the lumbar spine and control of lumbar lordosis (Macintosh & Bogduk, 1986). In contrast, the deep fibres are near the centre of lumbar vertebra rotation, and have a limited ability to assist in extending the spine (Panjabi *et al.*, 1989). The moment arm of this muscle is small, but it may exert its effects throughout the range of spine motion without compromise from its length-tension relation (McGill, 1991). Many of the trunk muscles are suited structure-wise to control the spine orientation, and most have a limited ability to control intervertebral shear and torsion (Panjabi *et al.*, 1989; Bogduk, 1997). The deep fibres of the multifidus are ideally placed to control these motions through intervertebral compression forces (Moseley *et al.*, 2002). The proximity of the deep multifidus to the centre of rotation means that it produces compression with minimal movement torque and thus minimal pressure on the spine (Panjabi *et al.*, 1989; Kaigle *et al.*, 1995).

It has also been reported that the control of shear forces and intersegmental motion by the deep fibres of the multifidus can occur irrespective of spine motion or internal and external forces (Moseley *et al.*, 2002). This has led to argument that the multifidus increases spinal stability and stiffness through tonic activity (Cholewicki *et al.*, 1997). Various states of activity have been suggested by various authors. Continued activity has been suggested by Valencia & Munro (1985) and Wolf *et al.* (1989). Donisch & Basmajian (1972) suggested phasic activity, and Morris *et al.* (1962) and Pauly (1966) suggested silent activity patterns.

Moseley *et al.* (2002: E34) reported that: *“One explanation for the functional distinction between the deep and superficial fibres of the multifidus may lie in the control of intervertebral shear and torsion through compressive force between segments. Because of their force vectors, the more superficial trunk muscles exert greater torque. If these muscles were recruited to control torsion and shear by increasing compression, then the component of their output that produces torque would need to be controlled. The resultant co-activation would result in an excessive energy and compressive ‘cost’. In contrast, the deep trunk muscles exert minimal*

*torque, which means that they can produce segmental compression with less resultant energy 'cost'."*

It is further suggested that the neuromuscular system can control individual segments of the spine, by a mechanism by which the segmental attachments of the deep multifidus provides flexibility (Panjabi *et al.*, 1989). The multifidus muscle is further able to support the spine by maintaining the alignment of the trunk against reactive forces that produces flexion by exhibiting anticipatory contractions during upper limb flexion, but not during extension. This provides evidence that the multifidus acts to control forces that attempts to create flexion rather than extension (Hodges & Richardson, 1996).

Another argument for multifidus as a postural control muscle can be related to the fibre type composition of both the deep and superficial multifidus, in that most of the fibres of the deep multifidus are of the slow-twitch fatigue resistant fibres (Sirca & Kostevc, 1985; Jorgensen *et al.*, 1993; Bajek *et al.*, 2000). The multifidus does contain a number of type 2a and 2b fibres that are smaller than the type 1 fibres. They are not resistant fatigue and are required for fast and power contractions. This greater number of fatigue resistant type 1 fibres has added to the suggestion of the multifidus as a muscle of postural control (Bajek *et al.*, 2000).

The multifidus has also been reported to poses a muscle spindle density that is quite low (Amonoo-Kuofi, 1983). It has been suggested that patients that have undergone surgery for disc herniation show definite changes in the multifidus muscle, which includes selective type 2 fibre atrophy, and internal structure abnormalities which are non-disease specific changes to type 1 fibre which appear 'moth-eaten'. This could be caused by such factors as muscle disease and disuse (Rantanen *et al.*, 1993).

This type of finding has added to the suggestion that type 2 fibres of the back muscles are exposed to insufficient workloads by people in a modern day setting, which is much more sedentary, and consequently the loads are insufficient to maintain the normal size and strength of the muscle fibres (Rantanen *et al.*, 1993). The fact that the type 2 fast twitch fibres have been found to be smaller and less in percentage has been suggested to be a result of modern lifestyles, in which powerful

contractions and heavy instant demands that would require fast twitch activation occurs less frequently to stimulation the retention of muscle size and strength (Bajek *et al.*, 2000). This has also lead to the suggestion that a higher intensity of exercise can be of benefit for those with low back pain (Mayer *et al.*, 1985).

Moseley *et al.* (2002) reported that the stability of the spine is challenged during arm movements and this causes different activations of the deep and superficial fibres of the multifidus. The superficial fibres seem to control spine orientation and the deep fibres control intersegmental motion, while the erector spinae group and the superficial and lateral fibres of the multifidus are activated earlier relative to movements such as deltoid flexion.

Moseley *et al.* (2002) reported that activity that is specific to direction is matched to the direction of reactive forces caused by limb movement and connected to the spinal orientation control (Cordo & Nashner, 1982; Aruin & Latash, 1995; Hodges & Richardson, 1997) and the displacement of the centre of mass (Aruin & Latash, 1995).

A non-direction specific pattern of activity is shown by the deep fibres of the multifidus and the transverses abdominis (Hodges & Richardson, 1997). The deep fibres of the multifidus can act in both directions with repeated arm movements, although force applications on the spine are in the opposite direction (Moseley *et al.*, 2002).

During repetitive movement about the 90 degree flexion, it seems that the deep multifidus becomes phasic. With control during reactive forces, it would appear that when the reactive forces are aligned more vertically, the contribution of the deep multifidus is reduced (Moseley *et al.*, 2002).

The multifidus is attributed to be more active during extension based movements and counteracting reactive forces in the sagittal plane when compared to the iliocostalis lumborum, which has the responsibility to regulate forces more in the frontal plane (Ng *et al.*, 1997). This has been confirmed during back extension

testing in which the multifidus will be more active during EMG extension testing and consequently shows higher rates of fatigue (Ng *et al.*, 1996; Ng *et al.*, 1997).

### 2.3.6.1.2 Transversus Abdominis

According to Kendall *et al.* (1993: 151):

Transversus abdominis

**“Origin:** *Inner surfaces of cartilages of lower six ribs, interdigitating with the diaphragm; thoracolumbar fascia; anterior three fourths of internal lip of iliac crest; and lateral one third of inguinal ligament.*

**Insertion:** *Linea alba by means of a broad aponeurosis, the pubic crest and pectin pubis.*

**Action:** *Acts like a girdle to flatten the abdominal wall and compress the abdominal viscera; upper portion helps to decrease the infrasternal angle of the ribs as in expiration. This muscle has no action in lateral trunk flexion except that it acts to compress the viscera and stabilize the linea alba, thereby permitting better action by anterolateral trunk muscles.”*

The contraction of the transversus abdominis causes an increase in intra-abdominal pressure and also causes tension generation in the thoracolumbar fascia. It has been reported in the past that this action causes a decrease in spinal loading by creating a trunk extensor moment (Grillner *et al.*, 1978; Gracovetsky *et al.*, 1985).

It has been speculated that contraction of the transversus abdominis could possibly enhance the stabilization potential of the spine, but there has been uncertainty of the direct mechanism involved (Tesh *et al.*, 1987; Cresswell *et al.*, 1992; McGill & Norman, 1993; Richardson *et al.*, 1999). There has been reported that the combination of transversus abdominis activation along with oblique activity will elevate intra-abdominal pressure and could as a consequence assist in spinal stabilization (Cresswell *et al.*, 1994).

Activation of the transversus abdominis can also act to stabilize the sacroiliac joint (Richardson *et al.*, 2002). Due to the corset-like structure of the transversus abdominis, the muscle can effectively flatten the abdominal wall and compress the viscera, which can lead to the anterolateral trunk muscles functioning more

effectively and to theoretically stabilize the spine (Farfan, 1973; McGill & Norman, 1993). This kind of functionality has made the transversus abdominis a focus of specific exercise training to manage back pain (Richardson *et al.*, 2002).

To be able to increase the stability of the spine to function during different postures and movements, the creation of a pressurised visceral cavity anterior to the spine can result in the production of a force against the apex of the lordotic curve in the lumbar spine, and assist in producing lumbar stability (Aspden, 1989). Also, translation and rotational motion of the spine can also be limited through lateral tension through the transverse processes.

Previous research has suggested that the activation of the transversus abdominis precedes external loads applied to the body and consequently it activates before the load is applied to the body. The transversus abdominis also activates before the other trunk muscles do (Cresswell *et al.*, 1994). Hodges and Richardson reported that the central nervous system initiates anticipatory contractions of the muscles of the trunk in expectance of possible movement and load application to the body.

Hodges and Richardson (1996) further reported that this type of anticipatory response due to possible external loading is more of a gross general muscular response. The body can only be sure of the magnitude and direction of the external load once it is applied to the body and then only apply the correct muscular response.

Displacement of the centre of mass occurs with limb movement and dynamic forces are transmitted to the body by the inertial reactions between segments (Friedli *et al.*, 1988). To be able to account for these changes the central nervous system needs to pre-programme the activation of the transversus abdominis and the other trunk muscles to act in anticipation of movement to precisely act to oppose the perturbing force acting on the body (Bouisset & Zattara, 1981). It has been reported that this type of pre-programming in activating postural muscles is activated as part of the motor command for movement of a limb or it is parallel with motor commands (Bouisset & Zattara, 1981; Horak *et al.*, 1984).

Rehabilitation has recently been focusing on the role of the transversus abdominis in the contribution to spinal stability. There are several muscles that have been identified to play a role in stabilization, but evidence has suggested the critical role of the transversus abdominis can thus have recommended more focus in a rehabilitation setting (Aspden, 1992; Cresswell *et al.*, 1994; Hodges & Richardson, 1996).

The transversus abdominis has been reported to be the primary muscle involved in creating and sustaining of intra-abdominal pressure, and has been established as the first of the stabilizing muscles to contract before movement of the body is initiated (Cresswell *et al.*, 1992; Cresswell & Thorstensson, 1994; Hodges & Richardson, 1996; Hodges & Richardson, 1997). Since the transversus abdominis contracts before the other stabilizing muscles, a delay in the activation of the transversus abdominis due to a deficit in motor control has been linked to those with low back pain (Hodges & Richardson, 1996).

Transversus abdominis has been found to contract prior to limb movement regardless of the direction of movement in those without low back pain (Hodges & Richardson, 1997), and it has been established that in those with low back pain, that the activation of transversus abdominis is delayed significantly (Hodges & Richardson, 1996). The effect of this decrease in motor control ability will be a lack of proper control of stability against forces acting on the spine and maintaining normal stability mechanics during normal movement (Hodges & Richardson, 1996; Richardson *et al.*, 2002).

#### **2.3.6.1.3 Internal and External Obliques**

According to Kendall *et al.* (1993: 148):

*“External Oblique, Anterior Fibres*

**Origin:** *External surface of ribs five through eight interdigitating with serratus anterior.*

**Insertion:** *Into a broad, flat aponeurosis, terminating in the linea alba, a tendinous raphe which extends from the xiphoid.*

**Action:** *Acting bilaterally, the anterior fibres flex the vertebral column approximating the thorax and pelvis anteriorly, support and compress the abdominal viscera,*



*depress the thorax and assist in respiration. Acting unilaterally with the anterior fibres of the internal oblique on the opposite side, the anterior fibres of the external oblique rotate the vertebral column, bringing the thorax forward (when the pelvis is fixed), or the pelvis backwards (when the thorax is fixed)."*

According to Kendall *et al.* (1993: 148):

*"External oblique, lateral fibres*

**Origin:** *External surface of ninth rib, interdigitating with the serratus anterior; and external surface of the 10<sup>th</sup>, 11<sup>th</sup> and 12<sup>th</sup> ribs, interdigitating with the latissimus dorsi.*

**Insertion:** *As the inguinal ligament, into anterior superior spine and pubic tubercle, and into the external lip of anterior one half of the iliac crest.*

**Action:** *Acting bilaterally, the lateral fibres of the external oblique flex the vertebral column, with major influence on the lumbar spine, tilting the pelvis posteriorly. Acting unilaterally with the lateral fibres of the internal oblique on the same side, these fibres of the external oblique laterally flex the vertebral column, approximating the thorax and iliac crest. These external oblique fibres also act with the internal oblique on the opposite side to rotate the vertebral column. "*

According to Kendall *et al.* (1993: 149):

*"Internal oblique lower anterior fibres*

**Origin:** *Lateral two thirds of the inguinal ligament, and short attachment on iliac crest near anterior superior spine.*

**Insertion:** *With transversus abdominis into crest of pubis, medial part of pectineal line, and into linea alba by means of an aponeurosis.*

**Action:** *The lower anterior fibres compress and support the lower abdominal viscera in conjunction with the transversus abdominis."*

According to Kendall *et al.* (1993: 149):

*"Internal oblique upper anterior fibres*

**Origin:** *Anterior one-third of intermediate line of the iliac crest.*

**Insertion:** *Linea alba by means of aponeurosis.*

**Action:** *Acting bilaterally, the upper anterior fibres flex the vertebral column, approximating the thorax and pelvis anteriorly, supporting and compress the abdominal viscera, depressing the thorax and assist in respiration. Acting*



*unilaterally, in conjunction with the anterior fibres of the external oblique on the opposite side, the upper anterior fibres of the internal oblique rotates the vertebral column, bringing the thorax backward (when the pelvis is fixed), or the pelvis forward (when the thorax is fixed)."*

According to Kendall *et al.* (1993: 149):

*"Internal oblique, lateral fibres*

***Origin:*** *Middle one third of the intermediate line of iliac crest and thoracolumbar fascia.*

***Insertion:*** *Inferior borders of 10<sup>th</sup>, 11<sup>th</sup> and 12<sup>th</sup> ribs and linea alba by means of aponeurosis.*

***Action:*** *Acting bilaterally, the lateral fibres flex the vertebral column, approximating the thorax and pelvis anteriorly, and depress the thorax. Acting unilaterally with the lateral fibres of the external oblique on the same side, these fibres of the internal oblique laterally flex the vertebral column, approximating the thorax and pelvis. These fibres also with the external oblique on the opposite side to rotate the vertebral column."*

Kendall *et al.* (1993) reported that significant weakness in the both the internal and external obliques can lead to functional as well as postural problems. Functional postural changes such as kyphosis, scoliosis and swayback postures along with changes such as an inability to flex the spine laterally as well as an anterior pelvic tilt in the standing position results due to a weakness in these muscles. Respiratory inefficiency and an inability to support the abdominal viscera can also result.

The obliques have also been identified as being important in the overall maintenance of spinal integrity. They provide lateral support and also play a role in stabilization by activating when the spine is placed under axial compression (Juker *et al.*, 1998). They have also been attributed to play a role in lateral bending and torso twisting (McGill, 1991).

In very similar fashion to the transversus abdominis, the internal and external obliques also seem to be able to anticipate upper limb movement and initiate contractions in advance of the movement (Belen'kii *et al.*, 1967; Bouisset & Zattara,

1981; Zattara & Bouisset, 1988; Aruin & Latash, 1995). Previous EMG research has reported that higher recruitment and co-activation of the internal and external oblique occurs during asymmetric tasks (Lavender *et al.*, 1992; Granata & Marras, 1993) such as combined sagittal and twisting motions. Activation seems to be less prominent when movement occurs in the midsagittal plane only (Lavender *et al.*, 1992; Granata & Marras, 1993; Granada & England, 2006).

It has been reported that an optimal movement strategy may allow variability in redundant, task-irrelevant dimensions, as in the kinematic variability in the transverse plane during midsagittal movement (Todorov & Jordan, 2002). Increased control is required during simultaneous movements such as combined movement in the sagittal and transverse planes, as this imposes higher levels of load on the body (Todorov & Jordan, 2002).

#### **2.3.6.1.4 Quadratus Lumborum**

According to Kendall *et al.* (1993: 143):

*“Quadratus lumborum*

**Origin:** *Iliolumbar ligament. Occasionally from upper borders of transverse processes of lower three or four lumbar vertebrae.*

**Insertion:** *Inferior border of last rib and transverse processes of upper four lumbar vertebrae.*

**Action:** *Assist in extension, laterally flexes the lumbar vertebral column, and depresses the last rib. Bilaterally, acting together with the diaphragm and fixes the last two ribs during respiration.”*

The quadratus lumborum can play a significant role in local lateral buttressing (McGill, 2002). It has been reported that the quadratus lumborum (QL) is a very effective lateral stabilizer due to its attachments to the lumbar vertebrae. During compressive loading on the spine, the first mode of buckling is lateral, and this will make the QL very effective in maintaining lateral stability of the spine (Lucas & Bresler, 1961; McGill, 2002).

To further support the idea of the QL being a stabilizer, it has been reported that the muscle seems to contract isometrically during spinal motion and hardly changes length during spinal motion (McGill, 1991; McGill, 2002).

#### **2.3.6.1.5 Erector Spinae (Extensor Group)**

The main muscles of the erector spinae group include the longissimus, iliocostalis lumborum and multifidus groups (Kendall *et al.*, 1993; McGill, 2002). However, due to the high importance of the multifidus, it was discussed in its own segment and will not be addressed here.

According to Kendall *et al.* (1993: 138):

*“Erector spinae group*

***Origin:*** *Common origin from anterior surface of broad tendon attached to the medial crest of sacrum, spinous processes of lumbar and 11<sup>th</sup> and 12<sup>th</sup> thoracic vertebrae, posterior part of medial lip of the iliac crest, supraspinous ligament and lateral crests of sacrum.*

***Insertion:*** *By tendons into the inferior borders of angles of lower six or seven ribs.”*

The pars thoracis components of these muscles attach to the ribs and vertebral components. They have relatively short contractile fibres with long tendons that run parallel to the spine to their origins on the posterior surface of the sacrum and medial border of the iliac crests (McGill, 2002). This group of muscles possess an effective moment arm to generate force due to the fibres being located underneath the fascia, and they thus produce an effective moment arm with the minimum amount of compressive force being subjected to the spine (McGill, 2002).

The lumbar and thoracic portions of these muscles have been partitioned in the past into the longissimus thoracis pars lumborum and pars thoracis, and into iliocostalis lumborum pars lumborum and pars thoracis (Bogduk, 1980). These two functional groups (pars lumborum, which attach to lumbar vertebrae and pars thoracis, which attach to thoracic vertebrae) show quite a number of differences. The lumbar and thoracic sections show differences in muscle fibre composition, in that the lumbar section shows an even distribution of type 1 and type 2 fibres, while the thoracic

section demonstrates a composition of about 75% slow twitch type 1 fibre dominance (Sirca & Kostevc, 1985).

#### 2.3.6.1.6 Gluteus Maximus, Gluteus Medius and Gluteus Minimus

According to Kendall *et al.* (1993: 226):

*“Gluteus maximus*

**Origin:** *Posterior gluteal line of ilium and portion of bone superior and posterior to it, posterior surface of lower part of sacrum, side of coccyx, aponeurosis of erector spinae, sacrotuberous ligament, and gluteal aponeurosis.*

**Insertion:** *Larger proximal portion and superficial fibres of distal portion of muscle into iliotibial tract of fascia lata. Deep fibres of distal portion into gluteal tuberosity of femur.*

**Action:** *Extends, laterally rotates, and lower fibres assists in adduction of the hip joint. The upper fibres assist in abduction. Through its insertion into the iliotibial tract, helps to stabilize the knee in extension.”*

According to Kendall *et al.* (1993: 221):

*“Gluteus medius*

**Origin:** *External surface of ilium between iliac crest and posterior gluteal line dorsally, and anterior gluteal line ventrally, gluteal aponeurosis.*

**Insertion:** *Oblique ridge on lateral surface of greater trochanter of femur.*

**Action:** *Abducts the hip joint. The anterior fibres rotate medially rotate and may assist in flexion of hip joint; the posterior fibres laterally rotate and may assist in extension.”*

According to Kendall *et al.* (1993: 220):

*“Gluteus minimus*

**Origin:** *External surface of ilium between anterior and inferior gluteal lines, and inferior gluteal lines, and margin of greater sciatic notch.*

**Insertion:** *Anterior border of greater trochanter of femur, and hip joint capsule.*

**Action:** *Abducts, medially rotates, and may assist in flexion of the hip joint.”*

### 2.3.6.1.7 Rotatores and Intertransversarii

The small rotatores muscles have been described as having a role of creating axial twisting torque, while the intertransversarii is thought to assist in lateral flexion. These muscles are very small and have small cross-sectional areas and moment arms, and their contribution to movement has been questioned due to their ability to generate only a few Newtons worth of force. It is believed that they could serve another function, possibly in assisting with stabilization rather than movement (McGill, 2002).

Some believe that these muscles could possibly serve as length transducers or vertebral position sensors in the spinal proprioception system. This is based on the evidence that these groups of muscles are well-supplied in muscle spindles, about 4.5-7.3 times the amount than the multifidus contains (Nitz & Peck, 1986; McGill, 2002).

### 2.3.6.1.8 Rectus Abdominis

According to Kendall *et al.* (1993: 147):

*“Rectus abdominis*

**Origin:** *Pubic crest and symphysis.*

**Insertion:** *Costal cartilages of the fifth, sixth and seventh ribs, and xiphoid process of sternum.*

**Action:** *Flexes the vertebral column by approximating the thorax and pelvis anteriorly. With the pelvis fixed, the thorax will move towards the pelvis; with the thorax fixed, the pelvis will move towards the thorax.”*

Due to the continuous ‘loop’ nature of the rectus abdominis, the muscle also serves to function as a transmission for lateral forces from the oblique muscles. Intermuscular tendons and fascia prevents the rectus from being torn apart by these lateral forces (Porterfield & DeRosa, 1998).

Kendall *et al.* (1993) suggested that weakness of this muscle will result in a decrease in the ability to flex the vertebral column. In the supine position it will be difficult to raise the shoulders and head off the group, while in the standing position, an anterior pelvic tilt will be permitted, which will increase the lumbar lordosis. A patient

presenting with these specific weaknesses may complain of pain across the low back. This is described as fatigue early on and later as an ache which may or may not progress to being acutely painful. Pain is usually worse at the end of the day and is relieved by recumbency such as a night's sleep (Kendall *et al.*, 1993).

In a very similar manner as the transversus abdominis, studies have demonstrated anticipatory contractions in the rectus abdominis and erector spinae before upper limb movement, especially before humeral flexion. This suggests that these muscles also function in an anticipatory manner towards stabilization (Friedli *et al.*, 1988; Zattara & Bouisset, 1988; Aruin & Latash, 1995).

On a side note, research has suggested that all sections of the rectus abdominis activate at similar levels during flexion torque generation. The so-called upper and low concept of rectus abdominis tends to function as one muscular unit, and the distinction between upper and lower rectus does not seem to exist (Lehman & McGill, 2001).

#### **2.3.6.1.9 Latissimus Dorsi**

According to Kendall *et al.* (1993: 279):

*“Latissimus dorsi*

**Origin:** *Spinous processes of last six thoracic vertebrae, last three or four ribs, thorough the thoracolumbar fascia from the lumbar and sacral vertebrae and posterior one third of external lip of the iliac crest, a slip from the inferior angle of the scapula.*

**Insertion:** *Intertubercular groove of the humerus.*

**Action:** *With the origin fixed, medially rotates, adducts, and extends the shoulder joint. By continued action, depresses the shoulder girdle, and assists in lateral flexion of the trunk. With the insertion, fixed assists in tilting the pelvis anteriorly and laterally. Acting bilaterally, this muscle assists in hyperextending the spine and anteriorly tilting the pelvis, or in flexing the spine, depending upon its relation to the axes of motion.*

Kendall *et al.* (1993: 279) further states: *“Weakness interferes with activities that involve adduction of the arm toward the body or the body toward the arm. The strength of lateral trunk flexion is diminished.”*

McGill (2002) adds that the latissimus dorsi has a potential lumbar stabilization function. Due to its origin at the lumbar spinous processes and its insertion on the humerus, it creates a lumbar extensor moment and stability, allowing it to be active during pulling and lifting motions, which has implications for how it is trained for functional motion patterns.

### **2.3.6.2 Neuromuscular Stabilization and Postural Control**

Biomechanical models have attempted to explain the contributions for various factors to the potential energy of the musculoskeletal system. These factors include muscle recruitment, spinal posture and external load (Bergmark, 1989a; Gardner-Morse *et al.*, 1995; Granata & Wilson, 2001). This is important because static stability is achieved when the equilibrium posture of the spine is also a state of minimum potential energy (Thompson & Hunt, 1984).

During static postural tasks the neuromuscular response to a kinematic perturbation will cause the system to return toward the equilibrium state (Peterka, 2003). Granata and Wilson (2006) stated that stability can be estimated from the time dependant behavior of kinematic variance. When a disturbance to the state of equilibrium has occurred, the neuromuscular control system seeks to maintain postural stability by actively working to return the system to an even equilibrium state (Peterka, 2003). A state of equilibrium can be observed when kinematics that has been measured are attracted towards the static posture equilibrium (Collins & DeLuca, 1993). The system will be returned to a normal state when a kinematic disturbance occurs during static postural tasks (Peterka, 2003).

Complex dynamic tasks are an effective way to indicate neuromuscular deficits in those with low back pain (Radebold *et al.*, 2001). It is assumed that the kinematics of each dynamic movement is the same as all the others during flexion-extension movements (Dingwell & Cusumano, 2000).

Granata and England (2006: E271) reported that: "*Kinematic variances about this target trajectory are the manifestations of stochastic disturbances and control errors during the movement process. Neuromuscular response to the kinematic*



*perturbations will cause the movement dynamics to be attracted towards the target trajectory*". Granata and England (2006) also report that when the sum of the exponents is negative, the system is stable. This is because the rate of expansion is lower than the rate of contraction.

The procedure of postural control is very complex, as it involves an integration of sensory and motor function (Leinonen *et al.*, 2003). This is the result of proprioception, which has been described as the sensation of the position, effort and movement at a joint which is associated with muscle contraction or the timing of muscle contraction, and is derived from receptors in muscles, joints and the skin (Gandevia *et al.*, 1992).

Receptors in various tissues play different roles in the proprioception depending on the position at a given joint (Swinkels & Dolan, 1998). Muscle spindles are believed to be activated through the whole physiological range of motion, while joint receptors are only believed to be activated near the end of the range of motion (Burgess *et al.*, 1992). Injury to a joint caused by instability in peripheral joints has been reported to be because of an inability of the joint to be accurately controlled due to a lack of position sense (Forwell & Carnahan, 1996).

This lack of proprioceptive control causes protective muscular contractions to occur too late because of a delay in neuromuscular reflex activation. This can then lead to excessive movement which will place abnormal loading on joint surfaces that can lead to pain and articular damage (Forwell & Carnahan, 1996). Injured joints have shown a reduced level of proprioception (O'Sullivan *et al.*, 2002).

It has been reported previously that lumbar stenosis patients tend to be more dependent on motor control rather than on sensory conduction between the lower limbs and the central nervous system during single leg standing tests (Leinonen *et al.*, 2002). During single leg standing tests, a greater dependency is observed to be on motor control than on sensory conduct in those with lumbar stenosis (Leinonen *et al.*, 2002). It seems that proximal body segments trigger postural reactions rather than lower leg proprioception (Bloem *et al.*, 2000).



Even before body disturbance occurs which are predictable, the body initiates protective lower limb muscle activations before the initiation of the disturbance occurs (Belen'kii *et al.*, 1967). It is therefore possible to assume that in addition to lower leg function, postural control seems to be related to information processing.

Feedback errors may result in the impairment of perception of lumbar movement resulting from sensory loss, deficits in information processing or a combination of these factors (Leinonen *et al.*, 2003). Muscle spindle activity has been reported to be of great importance, since a decrease in muscle spindle input has been found to decrease in those with lumbar pain. Muscle spindle input also seems to be important to ensure the correct positioning of the lumbosacral spine (Taimela *et al.*, 1999; Brumagne *et al.*, 2000).

It has been reported that soft tissue containing nociceptors and proprioceptors are injured in the process of sudden unexpected movements such as slips and falls due to an overreaction of the neuromuscular system (Lavender *et al.*, 1993). It has also been found that the generation of muscle force that is used to stabilize the spine is very often several times larger than the external load and the body weight combined (Radebold *et al.*, 2000).

Much larger forces placed on the spine is probably due to these much larger muscle forces that are responsible for most compressive and shear forces. Under static conditions, peak muscle forces tend to increase greatly under sudden loading conditions, and these forces are even more pronounced under extreme and sudden loading (Marras *et al.*, 1987; Lavender *et al.*, 1989; Lavender *et al.*, 1993).

It has been shown that the local muscle system is much more vulnerable to dysfunction due to chronic low back pain and instability due to neuromuscular system changes. Specific dysfunctions have been shown in the multifidus as well as in the deep abdominal muscles in those with chronic low back pain (Biedermann *et al.*, 1991; Lindgren *et al.*, 1993; Hides *et al.*, 1994; Hides *et al.*, 1996; Hodges & Richardson, 1996).

These types of changes seem to result in changes in the synergistic control between the different trunk muscles (Grabiner *et al.*, 1992; Edgerton *et al.*, 1996; Hodges & Richardson, 1996). It has consequently been found that in those with chronic low back pain, the global muscle system seems to substitute or even dominate over the impaired function of the local muscle system (O'Sullivan *et al.*, 1997).

Research has found that the requirement to control lateral flexion moment of the trunk in the direction of movement with upper limb abduction is accompanied by consistent early activation of the internal and external oblique, the rectus abdominis and the lumbar multifidus (Hodges & Richardson, 1996). This activation is not simply a general increase in background muscle activity, but is related specifically to an anticipated perturbation due to variation in time onset of each of the trunk muscles with different movement in various directions (Hodges & Richardson, 1996).

There are several factors that can contribute to this situation. More required neuromuscular effort is needed when moment increases with moment velocity to control and adjust kinematic disturbances (Granata & England, 2006). Along with an increase in trunk velocity and acceleration there is also an increase in torso muscle activity and co-contraction (Dolan & Adams, 1993; Marras & Mirka, 1993). Large motor unit activation is required to modulate the muscle forces when the activity of muscles is high, and this will automatically limit fine motor control during fast paced movement (Dolan & Adams, 1993; Marras & Mirka, 1993).

Fast dynamic movements reduce the time allowed for corrections by the neuromuscular system. This suggests an increased delay in the active requirement and neural feedback which is relative to the trajectory of the movement, and a delay in feedback is suggested as a factor that causes a decrease in stability in control systems (Ogata, 2002). It is suggested that higher kinematic errors may be expected when movement is fast, as suggested by the Fitt law of motor control (Fitts, 1954).

### **2.3.6.3 The Role and Application of Stabilisation**

Leinonen *et al.* (2003: 842) reported that: "*Protection from injury requires an ability of the body to anticipate events and to make suitable muscular responses. The appropriate proprioceptive information from trunk and lower limbs, as well as*

*functional motor control of the trunk and lower limbs is essential in the maintenance of postural stability”.*

Stabilization refers to the ability of the low back to maintain a position that is referred to as the neutral zone, and it has been suggested that those with low back pain demonstrates difficulty in achieving and maintaining this position for any length of time. This has been suggested to be caused by a possible discrepancy in proprioception (Lam *et al.*, 1989).

The position that is referred to as the position of neutral spine has been defined as a spinal position between end-range flexion and end-range extension (O’Sullivan *et al.*, 2003). A lack of neutral zone maintenance has been reported in those with a clinical diagnosis of lumbar segmental instability (Fritz *et al.*, 1998; O’Sullivan, 2000). A suggested dysfunction in stabilization muscles in the lumbar spine has led to the suggestion that this lack of position sense in the neutral zone of motion can lead to a problem in maintaining the neutral zone (Fritz *et al.*, 1998).

Stabilization has been suggested to be performed by two recognized techniques: bracing and the draw-in. Bracing involves a general isometric contraction of all the abdominal muscles, while the draw-in involves a more specific contraction of the transversus abdominis which involved the individual drawing in the abdominal wall independently of the other large trunk muscles (Richardson *et al.*, 2002). The drawing in maneuver is a more favoured technique used in lumbar stabilization exercise programmes (O’Sullivan *et al.*, 1998; Richardson *et al.*, 2002).

A contraction of the transversus abdominis has been demonstrated with the use of real-time ultrasound during a drawing in of the abdominal wall (Richardson *et al.*, 2002). Stabilizing of the trunk prior to limb movement is the goal of this technique, as it will cause an isolated contraction of the transversus abdominis as well as the multifidus, which will facilitate movement (Teyhen *et al.*, 2005).

The drawing in maneuver has been reported to activate the transversus abdominis preferentially to the internal and external obliques, which show little change with this

maneuver. This would seem to justify the use of this technique in the use of low back pain rehabilitation programmes (Teyhen *et al.*, 2005).

Also, during the draw-in pattern it has been reported that the multifidus muscle contracts along with the transversus abdominis (Richardson *et al.*, 1999). Richardson *et al.* (2002: 401) explained further that: “*The brace pattern was a general contraction of all the abdominal muscles, involving the individual performing an isometric bracing action. Real-time ultrasound imaging of a relaxed abdominal wall and during a brace of the abdominal wall demonstrates contraction of all the abdominal muscles. Surface EMG of both the oblique abdominal muscles and the erector spinae muscles demonstrated higher values during the abdominal bracing contraction than for the draw-in pattern.*”

It has been suggested that focus on minimal co-activation of the global muscle group and more focus on the isometric training of the deep abdominal muscles and multifidus should form the focus in the early stages of rehabilitation exercise programmes (O’Sullivan *et al.*, 1997). These contractions involve only a low level of the MVC (maximal voluntary contraction) are very specific and require a high level of patient compliance, but are very difficult to perform, due to the dominant substitution of other trunk synergistic muscles such as the rectus abdominis, external oblique, as well as the long back extensor muscles. The control of breathing also complicates the issue (O’Sullivan *et al.*, 1997).

During in vivo research, it has been suggested that only low levels of muscle contraction are required to achieve stability (Cholewickie & McGill, 1996). This is in line with the suggestion that strength training alone does not achieve proper motor learning and control, but depends more on patterning and inhibition of motor neurons. This will require selective inhibition of unnecessary muscular activity and the activation of additional motor units, which has to become a skilled learned by the individual (Basmajian, 1977; Edgerton *et al.*, 1996).

It has been suggested that the deep multifidus should be contracted independently of the global muscle group, namely the internal and external obliques and the rectus abdominis muscles (Magnusson *et al.*, 1996). The external oblique is also activated

during many therapeutic exercises simultaneously with the paraspinal muscles at the L5 level. Thus, it can be concluded that it is difficult to contract the lumbar paraspinal muscles (local stabilisers) independently from the external oblique (global stabilisers) during therapeutic exercises, as shown by surface EMG measurements (Arokoski *et al.*, 2004). However, in general, the activity of the abdominal muscles, especially the rectus abdominis, is lower than in the paraspinal muscles. This is an indication that load is mostly targeted at the paraspinal muscles during therapeutic exercises (Arokoski *et al.*, 2004).

Whole body exercise programmes are not recommended initially for those with low back pain, but exercises should rather focus on the activating of the transversus abdominis by means of precise self-bracing (using the drawing-in technique) techniques, which should occur independently of the other global abdominal muscles (Richardson *et al.*, 2002; Rasmussen-Barr *et al.*, 2009).

Other researchers have also advocated the training of muscular stabilization of the spine (Saal & Saal, 1989; Jull & Richardson, 1994). Thus, rehabilitation focuses on preferential activation of the deep trunk muscles (transversus abdominis and multifidus) during active movement. This has been theorised to improve the stability of the lumbar spine and found to decrease symptoms associated with low back pain significantly (Saal, 1990; O'Sullivan *et al.*, 1997; Danneels *et al.*, 2001; Richardson *et al.*, 2002).

## **2.4 Recommended Treatment Modalities for Low Back Pain**

Both pharmacologic and conservative treatments exist as safe and effective treatments for low back pain (Joranson *et al.*, 2002). Conservative treatment remains the preferred method when treating those with low back pain (Shirado *et al.*, 2005). Low back pain has been shown to be different in each case, and it is thus impossible to expect that all will benefit from a single treatment procedure only, and the success in treating low back pain is to identify different subgroups of low back pain patients of having a high probability of achieving success with specific interventions (Cleland *et al.*, 2005). A treatment that recognizes the non-medical factors involved in back pain has been called for, such as a biopsychosocial approach to low back pain (Fordyce, 1995).

Returning patients to normal levels of activity and reassuring them on the necessity of this has been suggested to be more of a priority for primary care physicians than only focusing on diagnostic studies and specialty referrals in improving the situation of those with low back pain (Waddell, 1996). Better long-term outcomes have not been achieved by repeat visits, diagnostic testing and specialist referrals but have added more to the cost of care than achieving better results (Sundararajan *et al.*, 1998; Carey *et al.*, 1999).

Further episodes of back pain incidences, occurrences, duration and work absence due to back pain can reportedly be prevented by means of physical exercise (Burton, 2005). Several guidelines recommend exercise therapy for chronic low back pain (Spitzer *et al.*, 1987; Albright, 2001; Hayden *et al.*, 2005; Krismer & Van Tulder, 2007). Chronic low back pain seems to share a close relationship with impaired trunk muscle function (Shirado *et al.*, 1992; Shirado *et al.*, 1995a; Ito *et al.*, 1996). To strengthen and improve flexibility is the main purpose of therapeutic exercise programmes (Shirado *et al.*, 1995b).

Stabilization training is used extensively in the rehabilitation of low back pain, and this type of training is different from general exercises by being more body specific and requiring more attention and precision from the patient involved (Bergmark, 1989a). Stabilization has shown to be very effective for low back pain, and it is because of this that it has been recommended that treatments for low back pain must be scientifically proven of its effectiveness (Richardson *et al.*, 2002).

#### **2.4.1 Acute Low Back Pain and its Necessity for Exercise Treatment**

Of all the cases of acute low back pain, about 80-90% of these cases will recover within 2-6 weeks of onset with or without treatment (Kendall *et al.*, 1993; Wright *et al.*, 2005). It has been reported that most patients with acute low back pain improve rapidly over a period of one month in terms of pain reduction, disability and return to work status, and after three months, all variables reaches a plateau and remains constant over a 1 year period (Pengel *et al.*, 2003).

Approximately 30% of those with low back pain in the primary health setting are pain free after one month and 60% are pain free after their first consultation. But, it has been reported that about 60% will experience one or more recurrent episodes in the subsequent year and 20-25% will still report a significant impact on their functional status due to low back pain (Von Korff & Saunders, 1996; Van den Hoogen *et al.*, 1997; Croft *et al.*, 1998).

When patients do recover spontaneous it is less likely that they will require treatment. Treatment might not improve the possibility of a successful outcome and might even cause a worsening of the situation by prolonging medical consumption, the duration of the disease and disability (Faas *et al.*, 1993; Sinclair *et al.*, 1997). Activities will be limited to a large degree, but this restriction should not be guided by pain but rather by time, and early return to activities is recommended (Simmonds & Dreisinger, 2003).

It has been suggested that an active rehabilitation programme has to be started as soon as possible (Wright *et al.*, 2005). Mayer *et al.* (2005) recommend a combination of direction-specific exercises along with a low-level heat wrap for return to functional activities. Recommendations state that exercise intervention programmes have to be started in the so-called sub-acute stage of low back pain, which is about between four weeks to three months. The expected spontaneous recovery of acute low back pain and the early intervention have to be taken into account during the acute phase of low back pain (Elders *et al.*, 2000; Karjalainen *et al.*, 2003b; Staal *et al.*, 2003; Hlobil *et al.*, 2005).

Low back pain can also be caused by muscle spasms. Kendall *et al.* (1993: 333) reported that: "*Muscle spasm is an involuntary contractions of a muscle or of a segment within a muscle that occurs as a result of painful nerve stimulation. Irritation from root, plexus or peripheral nerve branch level will tend to cause spasm of a number of muscles, while spasm due to irritation of the nerve endings within a muscle may be limited to the muscle involved, or may be widespread due to reflex pain mechanisms.*"



Kendall *et al.* (1993) further reported that protective spasm may occur secondary to injury of underlying structures such as ligament or bone. It then acts as a protection mechanism often following a back injury. This prevents movement and further irritation of the injured structure. The treatment of these types of reactive muscle spasms should ideally form part of any treatment programme.

The summary of the recommendation comes down to the guidelines that recommend that when a person first represents with acute low back pain, they first have to be examined for the so-called 'red flags', which are indications of serious underlying pathology (Koes *et al.*, 2001). These guidelines for red flags include weakness, particularly if localized in one area such as the leg; pain and/or difficulty controlling the bladder; numbness or tingling in the feet, legs or groin; severe, disabling or night pain; serious pain and a history of cancer or intravenous drug use; pain that does not subside within a couple of days; pain in the abdomen, as well as fever and weight loss along with back pain (Burton *et al.*, 2004). If the patient doesn't present with any red flags, current recommendations state that they should be advised to continue or gradually resume their activities of daily living (Waddell *et al.*, 1997; Koes *et al.*, 2001; Waddell, 2004).

Beyond this, further recommendations state that treatment has to be delayed until the patient has been away from work for at least 4-6 weeks. This is only to prevent the slip into chronicity, as many patients will recover spontaneously from an episode of acute low back pain (Frank *et al.*, 1996).

#### **2.4.2 Recommended Treatment for Chronic Low Back Pain**

Most other treatments for chronic low back pain have been reported to be only moderate in effect (Bogduk, 2004). One of the most effective and most recommended treatments for chronic low back pain is exercise (Koes *et al.*, 1991; Nordin & Campello, 1999; Van der Velde & Mierau, 2000; Friedrich *et al.*, 2005). The only problem is that the effects of exercise programmes have sometimes reported to be small and no form have been reported to be supreme over others (Van Tulder *et al.*, 2000; Arokoski *et al.*, 2004; Liddle *et al.*, 2004; Hayden *et al.*, 2005).



Several factors determine the success of therapeutic exercise programmes. Suggestions have indicated that exercise needs to be designed for the type and stage of the particular disorder to be successful (Nuvuaga & Nwuga, 1985; Deyo *et al.*, 1990; Graves *et al.*, 1990; Manniche *et al.*, 1991; Erhard *et al.*, 1994). The intensity and the execution of the technique have to be correct as well (Mitchell & Carmen, 1990; Kohles *et al.*, 1990; Manniche *et al.*, 1991; Tucci *et al.*, 1992). Regular and consistent performance of the therapeutic exercises will ensure full benefits. All prescribed sessions have to be attended and exercise intensity has to be maintained in the form of a home programme when the active intervention has ended (Graves *et al.*, 1990; Stankovic & Johnell, 1990; Manniche *et al.*, 1993; Saur *et al.*, 1996).

No evidence currently supports the use of mode of exercise over another (Burton, 2005). This could in part be due to the natural histories of low back pain not being reported properly. Specific exercise modalities that have been scientifically validated to improve low back pain are difficult to establish, since there are differences in methodology of research that make specific regimens difficult to decide on (Simmonds & Dreisinger, 2003).

A large degree of heterogeneity in terms of content has been used in physical exercise interventions (Kool *et al.*, 2004). Poorly designed studies have also contributed to the effectiveness of the role that exercise plays in the prevention of low back injuries (Simmonds & Dreisinger, 2003).

The biomedical approach has always been the favoured approach when managing chronic low back pain (Meyer, 2007). This approach uses a two-point perspective plan when pain is present. The underlying pathology is identified and localized. Secondly, the pain is removed with an appropriate cure or remedy (Vlaeyen & Morley, 2005).

The biopsychosocial approach views pain as a dynamic interaction between physical, psychological and social factors. More realistic treatment goals for patients include:

- The reduction, mostly not elimination, of pain (decreased self-reported pain scores)
- Improvement in physical and social function such as increased range of motion, standing and walking
- Improvement of vocational/disability status such as return to work and start job training
- Improvement of general functional status such as increased activities of daily living, social recreational activities and domestic activities
- Improvement in mood and associated symptoms such as sleeping patterns
- Increased self-management of pain, and development of active coping style and self-management skills
- Reduction or elimination of opiate and sedative-hypnotic medications
- Reduction in utilization of medical services such as reduced medical procedures, inpatient admissions and outpatient visits
- Modifying sensory input by medications and/or therapeutic modalities
- Addressing misunderstandings about the meaning of pain and associated anxieties towards the pain

(Ashburn & Staats, 1999; Sanders *et al.*, 1999; Simmonds & Dreisinger, 2003; Sanders *et al.*, 2005).

The use of the multidisciplinary/interdisciplinary approach has been receiving increasing support. This treatment model uses multiple therapies in a coordinated manner which employs active interaction and a common philosophy that encourages active involvement from the patient in the pain rehabilitation programme (Ashburn & Rice, 1998; Ashburn & Staats, 1999; Karjalainen *et al.*, 2001; Karjalainen *et al.*, 2003a; Rome *et al.*, 2004).

Kääpä *et al.* (2006: 371) reported that the goal of these treatment programmes is to: *“...to provide accurate information about back pain, lend attitudes favourable towards self-care, reduce fears and worries, assist patients in developing personalised action plans to manage their back pain, and to improve functional outcomes. In brief, the aim of the multidisciplinary rehabilitation is to provide effective coping strategies*

*despite of persisting bothersome pain.*” The pain rehabilitation programme may be defined as rehabilitation which primarily includes the dimension of physical rehabilitation and at least one of the dimensions that includes either the psychological, social or occupational dimensions (Guzman *et al.*, 2001).

This mode of treatment has been shown to produce the best outcomes in those with chronic low back pain. It places emphasis on functional restoration and functional capacity and develops coping strategies (Linton *et al.*, 2000; Caraggee, 2005). This type of approach enables patients to cope in the long-term by providing them with a type of self-management strategy (Rasmussen-Barr *et al.*, 2009). It has been reported that it is essential to address the psychosocial factors when treating musculoskeletal dysfunctions, otherwise treatment will not be as effective (Geisser *et al.*, 2005).

It has been debated that during the treatment of chronic low back pain if the primary focus should be on the reduction of pain. Pain is unpleasant for the involved person, but other major consequences of chronic low back pain such as restricted functioning, disability and work absenteeism are also long-term problems that need addressing (Staal *et al.*, 2003; Staal *et al.*, 2005). Patients also have to be recommended to stay active as long as possible (Deyo, 1996; Buchbinder *et al.*, 2001).

It has been recommended that treatment shouldn't focus primarily on pain, but rather on the consequences of pain, such as a loss of function, physical inactivity and being absent from work (Staal *et al.*, 2005). These goals are considered more important to treat, rather than pain itself. It is recommended that the reduction of pain should not be regarded as a primary goal of treatment. These other goals should rather be actively pursued, even if not reduction in pain levels occur (Sullivan, 2004). It has been suggested that the relief in pain found in studies using exercise intervention might be the result of the natural course of low back pain and not the result of the treatment programme (Shirado *et al.*, 2005).

The relief from pain has been reported not to be necessary to return to work (Lindstrom *et al.*, 1995; Crombez *et al.*, 1999; Van Tulder *et al.*, 2000). Low back pain

has a high rate of recurrence, and a life free of pain might be an unrealistic expectation, which as to be communicated to the patient in a way that they understand (Staal *et al.*, 2005).

Recommendations from research suggests that current goals should also focus on the eliminating of the use of unproven technologies or non-indicated medication, because chronic pain patients are vulnerable and at an increased risk due to their condition. Any treatment should be as conservative as possible and has to try and protect them from dangerous side-effects (Sanders *et al.*, 2005). Professionals trained especially in pain rehabilitation and management need to provide services and co-ordinated care across the various disciplines in order to achieve as many treatment goals as possible. Wherever possible, interaction should also exist between professionals, patients and their families to provide a social support network for the patient (Sanders *et al.*, 2005).

Care based on patient conditions and needs in an outpatient setting is also supported by the academic literature (Sanders *et al.*, 2005). It is recommended that a total of 20 treatment sessions per patient with chronic pain be used to match the recommendations of outcome based treatment studies (Sanders & Brena, 1993).

Advice to resume activities of daily living and work are reported to be without additional risk for aggravating back problems, since it has been demonstrated that exercise does not adversely affect the spine (Staal *et al.*, 2005). Less work absence has been reported in those who are advised to resume activities as compared to controlled treatments (Indahl *et al.*, 1998; Hagen *et al.*, 2000).

Adverse effects on work retention or current episodes of back pain have been reported to not be aggravated by a return to work and an absence of work restrictions (Hall *et al.*, 1994; Hiebert *et al.*, 2003). With current evidence, it appears that a return to work and normal activities is not associated with an increased risk for further episodes of low back pain (Staal *et al.*, 2005).

Bed rest for low back pain has generated conflicting evidence, since it is often prescribed for low back pain. Current evidence suggests that bed rest should only be

used in case of severe acute low back pain, and for nothing more than two days. Bed rest is not recommended for chronic low back pain (Brodke & Ritter, 2005).

### **2.4.3 The Role of Exercise as a Treatment Modality**

For chronic low back pain, exercise has shown to be a beneficial form of treatment (Van Tulder *et al.*, 2004). Evidence suggests that prescribed exercise does not increase the return rate of back pain in those with a history of chronic low back pain (Staal *et al.*, 2005). A significant decrease has been shown in those with a history of recurrent low back pain when prescribed exercises has been used as treatment modality (Donchin *et al.*, 1990; Soukup *et al.*, 1999; Hides *et al.*, 2001). It has been shown that when a treatment programme is medically supervised, exercise is more effective than usual care (Hurwitz *et al.*, 2002).

In those who are working full-time with recurrent low back pain and disability, it has been reported that a reduction in short-term and long-term disability as well as a reduction in short-term pain can be achieved with remedial exercise programmes (Rasmussen-Barr *et al.*, 2009). When prescribed exercise has been used, no study has found an increase in frequency of back problems associated with exercise programmes, and other studies have found no effect on recurrence rates (Staal *et al.*, 2005).

During the treatment of chronic low back pain it has been stated that back training programmes are effective treatment for the reduction of disability and the improvement of physical function (Abenhaim *et al.*, 2000). Active physical rehabilitation is now extensively prescribed as a treatment for chronic low back pain (Arokoski *et al.*, 2004). Exercise therapy can reduce pain intensity, alleviate functional disability, and improve back extension strength and endurance (Manniche *et al.*, 1991; Taimela & Härkäpää, 1996; O'Sullivan *et al.*, 1997; Kankaanpää *et al.*, 1999).

Rainville *et al.* (2000) reported that exercise can have a multitude of beneficial effects. An altering of pain attitudes and beliefs as well as an improvement of pain intensity and disability through a desensitization of fears can concerns are possible psychological benefits. Therapeutic benefits include the improvement of physical

function that is impaired by chronic pain. The prevention of work related fatigue and muscle pain are important factors than needs to be prevented, and this can be achieved by sufficient levels of muscle strength and good physical capacity (Oldervoll *et al.*, 2001). Cognitive intervention and exercise seem to help patients overcome their psychological barriers to pain and be more physically active (Keller *et al.*, 2003) as well as having a positive effect on patients' ability to cope with pain (Arnold, 2008). It has also been recommended that exercise programmes should contain functional exercises in which both the local and global muscles work together (Bergmark, 1989a). This has been formerly described to be important in an exercise protocol (Kavcic *et al.*, 2004).

An increase in cross-sectional area along with increases in muscle strength has been previously reported in stabilising muscles (Parkkola *et al.*, 1992; Takemasa *et al.*, 1995; Mannion *et al.*, 2001a). Exercise therapy has been shown to be more effective than general practitioners providing usual care. Exercise therapy and conventional physiotherapy (a combination of hot packs, massage, traction, mobilisation, short-wave diathermy, ultrasound, stretching, flexibility and coordination exercises, and electrotherapy) are equally effective for the treatment of chronic low back pain (Van Tulder *et al.*, 2000).

Irrespective of the type of exercise compared, studies have reported that exercise programmes containing active exercises are equally effective in those with chronic low back pain (Bentsen *et al.*, 1997; Ljunggren *et al.*, 1997; Mannion *et al.*, 1999; Bendix *et al.*, 2000). The effect of controlling negative pain behaviour relating to chronic back pain by exercise treatment is that intensive exercise programmes manages to make the patient expand the limits of their physical functioning and thus provides them with a feeling of pain control (Petersen *et al.*, 2002).

A decrease in the repeat of low back pain episodes by primary medical intervention doesn't seem to be sufficient. Results from long-term studies suggest that exercise treatment that is specific to low back pain and the recommencement of everyday activities has suggested to be more effective than medical treatment alone (Hides *et al.*, 2001). However, there is considerable variation in active physical treatment programmes for low back pain patients, both with respect to their duration and their

physical intensity. There also appears to be no direct dose-response relationship (Arokoski *et al.*, 2004).

Research findings indicate that the focus of therapy should be on helping patients learn awareness of body mechanics and dynamic posture; initiation and activation of a long-term exercise programme to gradually increase fitness, strength, coordination, a range of flexibility and motion; postural and muscle balance; specific physical coping strategies, as well as preventing debilitation caused by inactivity (Harris & Susman, 2002; Simmonds & Dreisinger, 2003; Liddle *et al.*, 2004).

Treatment modalities such as transcutaneous electrical nerve stimulation (TENS), ultrasound, heat and ice are regarded as secondary treatment options for those with chronic low back pain, and these should only be used if they assist in the ability of the patient to increase fitness, strength and range of motion (Schonstein *et al.*, 2003; Sluka & Walsh, 2003; Jousset *et al.*, 2004). Independently applied exercise and physical management programmes have to be set as long-term goals for the patient to be able to do when the active treatment has been successfully completed (Sanders *et al.*, 2005).

It has been suggested that the focus of exercise treatment in those with chronic low back pain should be on the local muscle system and the performing of specific stabilizing exercises (O'Sullivan *et al.*, 1997). The rationale for performing specific stabilizing exercises is that the repeated voluntary activation of the specific muscles induces plastic changes in the nervous system. This leads to a modification of the automatic recruitment of the trained muscle while performing functional tasks (Van Vliet & Heneghan, 2006; Tsao & Hodges, 2008).

It has been reported that beneficial effects in relieving pain and disability in those with chronic low back pain and decreasing recurrence rate after acute episodes has been achieved by performing exercises that promotes the independent contraction of the transversely orientated abdominal muscles along with the multifidus (O'Sullivan *et al.*, 1997; Richardson *et al.*, 1999; Hides *et al.*, 2001). Unfortunately, the size of the cross-sectional area of the multifidus muscle is not influenced by stability exercises and much more intensive functional exercises are needed to restore the



size of the multifidus muscle in those with chronic low back pain (Danneels *et al.*, 2001). This adds to the hypothesis that more intensive exercises are needed to restore the stabilisation muscles.

It has been previously reported that reductions in feed-forward control mechanisms of the trunk muscles have been observed in those with chronic low back pain (Hodges, 2001; Leinonen *et al.*, 2001). Observations in patients with low back pain have shown abdominal trunk muscle activation during upper (Hodges & Richardson, 1996; Hodges & Richardson, 1999) and lower limb movements (Hodges & Richardson, 1998). These muscle activations have also been observed during expected and unexpected upper limb and trunk loading movements (Magnusson *et al.*, 1996; Wilder *et al.*, 1996; Radebold *et al.*, 2000; Radebold *et al.*, 2001).

These functions appear to be improvable with active rehabilitation (Luoto *et al.*, 1996; Magnusson *et al.*, 1996; Wilder *et al.*, 1996). General exercises and advice to stay active have been shown to be beneficial for those with chronic low back pain (Maher *et al.*, 1999). In recent times, more specifically directed exercises for the spinal muscles in addition to general exercises have been recommended for those with chronic low back pain (Richardson *et al.*, 2002). Muscles that are associated with lumbar-pelvic stability have been targeted more frequently in with the aim of developing more effective and efficient exercise programmes for low back pain (Richardson *et al.*, 1999).

In conclusion it can be said that specific trunk muscle exercise programmes are aimed at restoring the structural and functional impairments that result from the effects of chronic low back pain (Kaser *et al.*, 2001; Mannion *et al.*, 2001a).

#### **2.4.4 The Use of Exercise Intervention in Chronic Low Back Pain**

A gain in muscle strength due to neural drive improvement will be the first effect of an exercise programme (Komi, 1986; Frontera *et al.*, 1988; Jones *et al.*, 1989; McCartney *et al.*, 1995). Next, hypertrophy of the muscle fibres will occur due to an increase in density (Jones *et al.*, 1989; Kadi, 2000). This will be followed by an increase in the cross-sectional area of the muscle (Keller *et al.*, 2003). In patients with chronic low back pain, selective muscle hypertrophy will occur after three



months of strength training. Research reports increases of Type 2 fibres in the multifidus with no change observed in the size of the Type 1 fibres (Rissanen *et al.*, 1995).

Pain may be moderated because of a relative reduction of physical load at work because of improved muscle strength (Oldervoll *et al.*, 2001). Muscle strength may increase with strength training, but it has been reported that at the L3-L4 level, the cross-sectional area and density remains the same (Parkkola *et al.*, 1992; Mannion *et al.*, 2000; Danneels *et al.*, 2001; Keller *et al.*, 2003). Density increases have been reported to be as much as 13% at the T12-L1 level, but the cross-sectional area in these muscles remains the same after training (Frontera *et al.*, 1988).

It is not clear whether specific modes of exercise, such as flexion, extension or strength training exercises are more effective than another (Van Tulder *et al.* 2000). The typical exercises that are tested and recommended by research include a combination of stretching, strengthening and unloaded movement exercises (Slade & Keating, 2006). Some studies uses home exercises along with formal supervised exercises programmes (Arokoski *et al.*, 2004). The present study did not use any form of unsupervised home exercises. Arokoski *et al.* (2004) reported that subjects in their study were given exercises to perform on their own at their homes. They found that most subjects exercised insufficiently at home when not being supervised.

It has been shown that the reduction of pain and disability during active physical rehabilitation is strongly dependent on a decrease in psychological distress and fear avoidance (Mannion *et al.*, 2001b). Even though pain and fear avoidance behaviours were addressed by explaining and motivating the subjects, there was still a lack of compliance from the subjects to perform the exercises at home (Arokoski *et al.*, 2004). Rasmussen-Barr *et al.* (2009) found no improvement in fear avoidance even when exercise treatments were supervised.

It has been suggested that the lumbar paraspinal muscles are both aerobic and anaerobic during therapeutic exercises when measured using surface EMG (Åstrand & Rodahl, 1991). The highest level of paraspinal electromyographic activity has been reported to be during exercises that involves lifting the hips up to a bridge position

when supine and during bilateral leg extensions in the prone position. The same authors found that exercises involving hyperextension of the back from the prone position are not the only exercises that can activate the lumbar paraspinal muscles (Arokoski *et al.*, 2004). An increase in muscle activity is produced when extra load is generated, such as holding additional weights or unbalanced limb movements (Arokoski *et al.*, 1999; Arokoski *et al.*, 2001).

On its' own, low back pain will not affect the exercise response, but sitting or standing positions may worsen pain and the patient may be prevented from performing at recommended exercise intensities or even cause a variation in effort. Patients should thus perform a variety of exercises in different positions and limitations should be identified as soon as possible (Simmonds & Dreisinger, 2003).

Petersen *et al.* (2002) compared McKenzie training and intensive strengthening exercises on chronic low back pain subjects in an outpatient-based clinic. The McKenzie group received standard McKenzie-based therapy, while the strengthening group performed their exercises in a group setting while being supervised, consisting of six subjects at a time (Petersen *et al.*, 2002).

A session began with 5-10 minutes on a stationary cycle succeeded by low-intensity warm-up exercises for 10 minutes of 10 repetitions of low resistance exercises for the lumbopelvic muscles in flexion, extension and rotation. This was then followed by intensive dynamic strengthening training that was performed in flexion and extension (Petersen *et al.*, 2002). The authors chose this type of training because it was shown to be effective in the treatment of chronic low back pain, as conducted by Manniche *et al.* (1988). Repetitions were progressed and the programme was conducted for eight weeks with two sessions per week. The authors showed improvements in both groups, but no statistically significant difference between the two groups (Petersen *et al.*, 2002).

Arokoski *et al.* (2004) reported that as measured by surface EMG, a prolonged holding of the paraspinal muscles during certain exercises appears to sufficiently activate these muscles in the re-education phase. They also found that certain exercises should be added later in the rehabilitation programme when greater muscle loads

can then be tolerated. In order not to risk further injury, exercises that cause the least amount of strain should be done in the beginning of the programme (Arokoski *et al.*, 2004).

It has been suggested that training the lumbar muscles for endurance by means of longer programmes and lower effort seem much more preferable over pure strength and power training (Biering-Sorensen, 1984; Luoto *et al.*, 1995; McGill, 1998; McGill, 2002). Dynamic endurance training should be encouraged over static endurance training (Moffroid, 1997).

Sherman *et al.* (2005) compared the effect of yoga, exercise and just reading a self-care booklet to establish which was more effective. Their study lasted for 12 weeks and subjects attended weekly supervised classes, as well as exercising at home unsupervised. Their yoga was a traditional style in which the exercises were designed to be safe for those with low back pain.

Their exercise group included strengthening exercises for leg, hip, abdominal and back muscles. These were increased in terms of repetitions performed over the course of 12 weeks (Sherman *et al.*, 2005). Both the yoga and exercise groups performed their programmes for 75 minutes at a time. The study reported that the yoga group reported superior outcomes compared to the exercise group, but these were neither statistically nor clinically significant. The authors reported that yoga exercise might be beneficial for those with chronic low back pain, not because of exercise features alone, but rather through its benefits of linking physical movements with mental focus.

From a physical perspective, popular lore persists that yoga increases flexibility and strength, tones muscles and releases muscle tension (Sherman *et al.*, 2005). Several studies of patients with low back pain found that yoga increased hip flexion (Williams *et al.*, 2003), and spinal and hamstring flexibility (Baldwin, 1999; Galantino *et al.*, 2004). The authors of the study consider their form of yoga safe for persons with chronic low back pain (Sherman *et al.*, 2005).

It has been suggested that a concern for the use of exercise as a treatment would probably manifest itself in the long-term, and not in the form of any short-term adverse effects (Staal *et al.*, 2005). However, research has suggested that lower rate of recurrence of low back pain and reduced work absenteeism has been reported where regular exercise habits have been followed over a 14-month period, with regards to prescribed exercise regimens compared to those who show poor compliance (Taimela *et al.*, 2000).

As has been reported, exercise remains an effective method for treating chronic low back pain, but compliance to maintain a prescribed regiment of regular therapeutic exercise has been problematic, and has been reported by several authors (Martin *et al.*, 1984; Reilly *et al.*, 1989; Sluijs & Knibbe, 1991). Research reported has suggested that up to two-thirds of patients show poor compliance with exercise, which is especially relevant with unsupervised home training (Reilly *et al.*, 1989; Sluijs & Knibbe, 1991; Sluijs *et al.*, 1993; Nelson *et al.*, 1995).

It has been reported by Oldervoll *et al.* (2001) that the attendance rate in their research was 77% in their strength promotion group and 81% in their endurance training group. Grønningsæter *et al.* (1992) reported an attendance rate of 80% among women and 76% among men. Their participants were offered training during paid working hours. The training in the Oldervoll *et al.* (2001) study took place just before or after work hours, being very similar to the present study.

It has been reported that attendance for exercise sessions seems to be more related to intrinsic motivation factors rather than exercise sessions taking place within or outside of working hours (Oldervoll *et al.*, 2001). According to Robinson & Rodgers (1994) the completion of training depends on physical factors such as motivation, education and knowledge of and belief in the beneficial effects of physical activity on health, weight and mental health.

It has been suggested however, that clinical outcome is not necessarily associated with exercise compliance (Sluijs *et al.*, 1993). Observed result seems to suggest that valid objective adherence protocols towards exercise recommendations just simply do not exist (Friedrich *et al.*, 2005). Valid and reliable tools to assess the degree of

patient compliance has been shown to be lacking by several authors (Deyo, 1982; Faas *et al.*, 1995; Pfingsten *et al.*, 1997). Overstating of compliance and not reporting noncompliance can be attributing factors to patients performing worse on their outcome (Friedrich *et al.*, 2005).

In summary, dropout rates seem to be high in exercise intervention studies. Geisser *et al.* (2005) reported a dropout rate of 28% and Koes *et al.* (1996) reports dropout rates of greater than 20%. Petersen *et al.* (2002) reported a dropout rate of 30%. Past research has indicated that there is a high rate of non-compliance with exercise. Around 50% of subjects in supervised studies will drop-out within six months (Dishman, 1991). It has been reported that this phenomenon is not uncommon in studies of chronic low back pain patients even in an outpatient setting (Bentsen *et al.*, 1997; Keel *et al.*, 1998; Snook *et al.*, 1998).

#### **2.4.5 Conservative vs. Aggressive Exercise Treatments**

Conservative rehabilitation programmes for low back pain have always been effective. For example, previous authors had subjects performing gentle co-activation exercises of the multifidus and transverses abdominis muscles with real-time ultrasound feedback imaging. These subjects had significantly fewer recurrences than those performing no exercise (Richardson *et al.*, 1999; Hides *et al.*, 2001).

Pain and disability measurements show to be stable over a one-year period of time after an aggressive exercise-based rehabilitation programme was completed and high levels of compliance was shown with recommended exercises following the intervention programme (Hartigan *et al.*, 2000). Intensive exercise programmes have also shown to have large effect on short-term pain and function as compared to other treatments (Manniche *et al.*, 1991; Johannsen *et al.*, 1995; Petersen *et al.*, 2002). Ostelo *et al.* (2003) also reported that intensive exercise programmes were more effective on functional status and faster return to work in first-time lumbar disc surgery patients.

Exercise does seem to affect the spine in any sort of negative way. Even the aggressive and more intensive exercises performed by elite athletes shown no more negative effects than in non-athletes. Low back pain seems to occur less frequently in athletes and no greater frequency of sciatica is reported in athletes as compared to healthy controls (Videman *et al.*, 1995).

#### **2.4.5.1 Does Aggressive Exercise Rehabilitation Play a Role in Managing Chronic Low Back Pain?**

The issue of more aggressive exercise regimes for the treatment of chronic low back pain remains controversial. Goldby *et al.* (2006) reported that rehabilitation programmes show efficacy in patients with chronic low back pain but they often include universal aerobic or strenuous exercise regimens (Van Tulder *et al.*, 1997; Maher *et al.*, 1999; Van Tulder *et al.*, 2000; Furlan *et al.*, 2001; Mior, 2001).

Little importance is attached to the use of aerobic capacity in itself for the management of musculoskeletal pain (Grønningsæter *et al.*, 1992). A combination of aerobic exercises along with strength developing activities is used in most physical exercise intervention studies, but bias has been shown towards aerobic activities and thus the relative importance regarding the use of these two regimens remains unclear (Oldervoll, 2001). Increased aerobic activity has not shown to be a crucial mechanism in the reduction of low back pain (Oldervoll, 2001). Goldby *et al.* (2006) used exercises for only four muscles and focused on implementing the contractions achieved by the exercises into everyday postures and positions. Significant improvements with this regime from pre-test to post-test were shown in their research. The results were ascribed to 'immeasurable physiological effects', as well as factors such as peer support, patient empowerment and self-treatment, which has also been described by Long *et al.* (1996).

### **2.5 Ergonomics: The Key to Protecting the Spine**

Mechanical circumstances that have been identified as key factors in causing low back injuries are sudden loading incidents such as trips, slips, falls and bending and twisting while lifting (Frymoyer *et al.*, 1983; Kelsey *et al.*, 1984; Bigos *et al.*, 1986; Omino & Hayashi, 1992). These types of incidences accidentally arise during recreational activities. A higher rate however has been identified among professional

personnel such as nurses handling patients. Most of them will recover with six weeks of an acute injury but some may become chronic (Radebold *et al.*, 2000).

### **2.5.1 The Role of Ergonomic Modification and Risk Factor Prevention**

The concept of risk prevention is poorly understood and documented inconsistently, but it is important and highly relevant to understand the relationship (Burton, 2005). A previous history of low back pain has been identified as being the most powerful indicator of future episodes of low back pain (Hestbaek *et al.*, 2003b).

Other important factors that have also been identified as being important indicators of risk are heavy physical work, frequent bending, twisting, lifting, pulling/pushing, repetitive work, static postures, vibrations and obesity, which stresses the disc endplates and facet joints. Smoking has also been identified as an important risk factor due to the reduced oxygen to the spinal structures (Andersson, 1997; Jansen *et al.*, 2002; Laursen & Scibye, 2002; Leboeuf-Yde, 2004).

Other factors such as rapid work pace, repetitive motion patterns, insufficient recovery time, heavy lifting, non-neutral body postures (either dynamic or static), mechanical pressure concentrations, vibration (both segmental and whole-body) and low temperatures have been identified as possible ergonomic risk factors in occupational settings that can be related to incidences of low back pain (Punnett *et al.*, 2005).

Rotation has been identified as having a greater risk factor than forward bending, and this risk increases when rotation is added to other postures (Prado-Leon *et al.*, 2005). A lack of activity has also been identified as being important, as an inactive spine or even an overactive spine performing high loads of physical activity are believed to be at a disadvantage. A U-shaped curve is believed to exist, where sedentary work and hard work are both perceived to be harmful. An occupation that requires some movement in combination with lighter tasks are believed to be better for the lumbar spine (Leboeuf-Yde, 2004). From a biological point of view, sufficient mechanical loading is needed to increase the strength of the soft tissue, but too much loading will result in tissue breakdown (McGill, 2002).



It has been suggested that static postures can contribute significantly to back pain and that the risk is associated with the maintaining of mild trunk flexion (between 21-45 degrees) (Punnett *et al.*, 1991). The risk is further increased when the trunk is twisted for more than 45 degrees or flexed laterally more than 20 degrees (Punnett *et al.*, 1991).

Prolonged seated postures especially followed by immediate lifting of heavy objects have also been identified as a risk factor (Van Vuuren *et al.*, 2005). Sitting for long periods of time also causes creeping changes in the posterior ligaments as well as in the position of the nucleus within the annulus (Adams & Hutton, 1988; McKenzie, 1979). It has been shown that only half of the intervertebral joint stiffness is regained in two minutes after 20 minutes of full flexion and some joint laxity remains after 30 minutes (McGill & Brown, 1992). It is thus considered that it is a risk factor for the development of low back pain in some occupation that lifting is required to be done with a so-called 'unstable back' (Van Vuuren *et al.*, 2005).

Low back injury risk is shown to be related to the dynamic rate of movement during repetitive trunk flexion (Marras *et al.*, 1995a). As reported earlier, evidence seems to suggest that when trunk movements include non-sagittal movement components, the risk of low back injury is further increased (Fathallah *et al.*, 1998). During fast paced movements, there is a higher spinal load applied due to the influence of muscle recruitment and co-contraction of the spinal muscles which causes a load higher than with slower movements (Granata & Marras, 1995).

Analysis of lifting exertions suggests that co-contraction may be recruited, in part, to augment spinal stability (Cholewicki *et al.*, 1997; Gardner-Morse & Stokes, 1998; Granata & Orishimo, 2001). Dynamic trunk flexion could carry the risk of reduced spinal stability with increased spinal compression during dynamic trunk flexion (Granata & England, 2006). Van Vuuren *et al.* (2005) reported no increased risk during bending only, but found a much higher risk when torso flexion and twisting are combined in an occupational setting.



Psychosocial risk indicators include distress, depression, beliefs, job dissatisfaction and mental stress at work (Andersson, 1997; Hoogendoorn *et al.*, 2000; Linton, 2000).

One of the potential risks of physical activity and exercise in cases of chronic low back pain might be that it could be counter-productive to increase physical activity during episodes of pain, as physical activity involves increased biomechanical loading, which might worsen the condition of damaged spinal structures (Staal *et al.*, 2005). There is inherent risk for back injury and pain with all human activities, including exercise and work.

For the development of back pain or disc degeneration, exercise and sports participation for those without low back pain has shown no major risk factors for the development of back disorders (Staal *et al.*, 2005). Studies involving children (Harreby *et al.*, 1997), college students (Cahmak *et al.*, 2004) and adults (Suni *et al.*, 1998; Croft *et al.*, 1999) have reported that regular exercise seems to maintain healthy back status and leads to lower risks for the development of new episodes.

Exercise and sports participation have been argued not to be significant risk factors for low back problems, as a lack of participation has been reported as a risk factor for the progression of lumbar disc degeneration (Elfering *et al.*, 2002). Even among workers with jobs that require lifting over 5 000 kg per shift personal fitness level and frequency of physical activities have a positive effect on reducing the incidence of back pain compared to co-worker with much lower activity and fitness levels (Stevenson *et al.*, 2001).

It has thus been reported that prescribed exercises for those with low back pain can be relatively safe without adding difficult to deal with risk for additional injury or pain since exercise does not seem to cause an increase in risk of back pain in the asymptomatic population if the exercises are prescribed correctly (Staal *et al.*, 2005).

### **2.5.2 Specific Task Modification: Occupational Risk Factor Management**

An occupationally related accident or activity is often blamed for damaging spinal structures and causing low back pain (Staal *et al.*, 2005). It is then when concern

becomes legitimate that prescribed physical demanding exercises, daily activities and work may cause further damage to the spine and lead to increased symptoms when the notion of cause and effect is extended into a medical setting and specific causes are explored. This could explain why restrictive recommendations for work and activities to those with chronic low back pain are provided by health care providers to manage pain and symptoms (Rainville *et al.*, 2000).

McGill (2002) recommends the following guidelines for the stages of patient progression for low back pain rehabilitation:

1. Identify and remove exacerbating activities.
2. Record in a journal the state of the low back throughout the day.
3. Develop spine position awareness.
4. Begin appropriate spine rehabilitation exercises and abdominal stabilisation.
5. Develop muscular endurance.
6. Transfer these to daily activities.

Ergonomic stressor prevention has shown good potential for disease reduction, but interventions into these types of interventions have not yet been widely implemented (Punnett *et al.*, 2005). The removal of these stressor has been hypothesized to remove back pain or at least reduce its effects (Frank *et al.*, 1996; Marras *et al.*, 2000). The major types of stressors that have been identified to be removed includes the redesign of workstations to eliminate the need for bending and twisting, installation of material or patient hoists and other lifting devices, greater variety of work tasks to avoid repetitive loading of the same body tissue and structure, and improved mechanical isolation to reduce whole-body vibration transmission (Frank *et al.*, 1996; Marras *et al.*, 2000).

### **2.5.3 The Back School Concept: The Role of Research and its Application**

The back school concept boils down to education. It can be described as a group intervention, conducted or supervised by a paramedical therapist or a medical specialist, consisting of both an educational/skills programme and exercise intervention programmes (Heymans *et al.*, 2004). The purpose of this type of programme is in educating the patient regarding the nature of the low back pain and

disorder, and also assists them to form positive and active attitudes as well as placing emphasis on correct body mechanics and partaking in prescribed physical exercises (Hall & Iceton, 1983; Cohen *et al.*, 1994; Turner, 1996).

For those suffering from chronic low back pain, back schools have been shown to be effective in the occupational setting (Tulder *et al.*, 2001). Friedrich *et al.* (2005) used an exercise programme in conjunction with a motivational programme to treat patients suffering from chronic low back pain. Their motivational programme included extensive counselling and information strategies (such as reinforcing the internal locus of control, patient problem solving, emphasising the importance of exercise), using positive reinforcement techniques when compliance was given to exercise, signing a contract agreeing to participate in the exercise programme as well as patients reporting on the exercises that they perform each day.

They found that the combination of exercise and the motivational programme was significantly more effective than exercise alone in decreasing pain and disability, and increasing the degree of working ability (Friedrich *et al.*, 2005). This finding is explained by the effect on long-term success in that the combined exercise and motivational programme provides the patient with a ready set of tools that are retrievable even after treatment termination. It would support the patient in dealing with the multifaceted psychosocial phenomenon of chronic low back pain (Friedrich *et al.*, 2005).

It has to be explained to the patient that factors that maintain pain can be different from the factors that causes it, thus validating their pain (Meyer, 2007). For the general population, biopsychosocial principles from information and education should be given to the patient, for it has shown that it improves back beliefs and can have a positive influence on health and vocational outcomes (Burton, 2005). The intensity of the pain experienced by the patient may be increased by fear avoidance beliefs and catastrophising (Meyer, 2007). The beliefs that patients have about their fear of pain and injury must be targeted in the early phases of the pain development, for their beliefs are important factors contributing to long-term disability and work loss (Waddell *et al.*, 1993; Picavet *et al.*, 2002).

According to Meyer (2007) certain myths about chronic pain exist that have to be expelled as soon as possible. These myths include

- Search long enough, and you will find the cause and the cure.
- Abnormal scan results validate and explain the pain.
- Only organic pain is real.
- You have to learn to live with it.
- Let the pain be your guide – rest when it hurts.
- Pain is equal to tissue damage (McIndoe, 1994).

For patients with chronic pain, the need for behavioural and psychological treatment in the interdisciplinary rehabilitation setting has been backed up with strong evidence from the literature (Sanders *et al.*, 1999; Dworkin & Breitbart, 2004; Keefe *et al.*, 2004). A patient will need psychological/behavioural treatment if significant levels of depression and anxiety are present, along with pharmacological intervention for symptoms if needed. When present, psychological/psychiatric conditions such as post-traumatic stress disorder and social adjustment issues should also receive treatment they present symptoms (Sanders *et al.*, 2005). Access to stress management training, cognitive behavioural therapy, operant therapy and biofeedback should also be available as the condition of the patient requires it (Astin, 2004).

Low intensity ‘Swedish Back School’ principles have shown to not be as effective as higher intensity back schools in occupational settings (Heymans *et al.*, 2005). A reduction in pain and less frequent episodes have been demonstrated by higher intensity back schools (Heymans *et al.*, 2006).

The low intensity back school based on the Swedish principle is determined by its application. This usually consists of four group sessions once a week for four consecutive weeks (Heymans *et al.*, 2006). Each session is divided into an educational (30 minutes) and a practical part (90 minutes), and guided by written information and a standardised exercise programme (Heymans *et al.*, 2006).

Subjects receive information regarding coping with back pain in their work settings as well as the work setting itself. The exercise part comprises a standardised exercise programme of strength training and home exercises. This involves progressive resistance training as well as functional exercises (Heymans *et al.*, 2006).

High intensity back school in the Heymans *et al.* (2006) study was conducted twice a week over eight weeks. It consisted of 16 sessions, each lasting an hour where the principles of cognitive behavioural therapy were applied throughout the programme (Vlaeyen *et al.*, 1995). Work simulating and strength training exercises were performed during subsequent sessions with a gradual increase in resistance (Heymans *et al.*, 2006). However, it has been reported that workers treated in low intensity back school groups return to work faster and were absent from work for fewer days than compared to high intensity back schools (Indahl *et al.*, 1995; Indahl *et al.*, 1998; Heymans *et al.*, 2006).

A beneficial effect on work absence has been reported by others in the form of high intensity graded active intervention that contained high intensity back school, but effects have been reported as appearing slow (Staal *et al.*, 2004). It has been reported that for patients in an occupational setting that suffer from chronic low back pain, intensive intervention programmes show effective results (Guzman *et al.*, 2001; Schonstein *et al.*, 2003).

It is also recommended that high intensity back schools utilising both an educational/skills programme and exercise may be used for those patients with recurrent and persistent pain (Burton, 2005).

Goldby *et al.* (2006) used subjects suffering from chronic low back pain over a time period of 10 weeks and randomised them into three groups. Their first group received a stabilisation exercise programme; the second group manual physiotherapy and their third group only received education in the form of a back school. The latter was their control group. All three their groups received the back school. However, the control group showed the least improvement in overall scores

compared to the other groups. They showed significant improvement in pre-test scores.

It may then be argued that the back school is not effective only on its own; it needs to be combined with other treatment modalities to achieve an optimal effect.

## **2.6 Research Problem**

As stated by McGill (2002) most of the exercises prescribed for chronic low back pain are of single modal only and they have not assessed the impact of progressive treatment methods. Thus, research on this subject has been found to be limited.

Conservative treatment may be classified into three phases: primary, secondary and tertiary rehabilitation (Mayer *et al.*, 2001). The first phase is the acute management phase, which is treated for 0-12 weeks after onset and can be considered as the primary phase (Shirado *et al.*, 2005). Next, physical deconditioning has to be prevented by preventing chronic disability in the secondary phase of management. The tertiary phase involves the prevention of permanent disability for those who already suffer the effects from chronic disability by a full interdisciplinary team (Mayer *et al.*, 2005). Either secondary or tertiary rehabilitation should ideally be used on patients that present with chronic low back pain (Shirado *et al.*, 2005).

Irrespective of the type of exercises that are used, it has been shown that treatment programmes that contain active exercises are similarly effective in treating those with chronic low back pain, as shown by several studies. A method for controlling pain and inhibiting negative pain behaviour that is associated with pain is suggested to be in the form of intensive exercise programmes. These programmes are hypothesized to make the patients expand the limits of their physical functioning (Petersen *et al.*, 2002).

In a well-controlled randomised trial Petersen *et al.* (2002) compared the effect of McKenzie therapy to intensive strength training in the treatment of chronic low back pain. They concluded that the McKenzie method and intensive strength training seemed to be equally effective in the treatment of chronic low back pain.

It has been suggested however, that there exists too little research into popular exercise techniques like the McKenzie technique to validate its' use as a treatment option. It has thus been recommended that the optimal intensity, frequency, duration and specific types of exercises be further investigated to validate their use in the clinical setting as well as in the academic literature (Hildebrandt *et al.*, 2004).

A barrier has been reported in the accurate replication of many interventions in that programmes used in research have been described completely. To have the details that would enable replication of interventions used will advance the science of exercise prescription (Slade & Keating, 2006). Uncertainty exists whether anyone type of exercise regimen (such as flexion, extension, and/or strengthening exercises) is more effective than others (Van Tulder *et al.*, 2000).

The aim of this study will then be to solve the problem by comparing conservative treatment methods to more progressive-aggressive multimodal treatment methods in the form of remedial exercise along with cognitive behavioural techniques in the form of the back school approach. The aim of the study will then be to investigate the two forms of treatment methods in the form of remedial exercises along with cognitive behavioural techniques in the form of the back school approach and to then compare their effects.

A high intensity back school approach as well as a low intensity back school approach will be used for this study.

The student will attempt to answer the following questions through his research:

- How effective are progressive-aggressive exercises versus more traditional exercise in the treatment of chronic non-specific low back pain?
- What exercises will be effective and how effective will they be when progressed?
- How will a more aggressive approach influence the outcomes compared to the more traditional approach to remedial exercise therapy?



## CHAPTER 3

### METHODOLOGY

#### 3.1 Introduction

The focus of the present study was to test low back muscle strength as well as psychological factors in subjects with chronic low back pain, place them on two exercise intervention programmes (conservative or progressive-aggressive programmes) for 12 weeks, and re-evaluate them according to the original protocol. Psychological factors were also tested at week 4 and week 8. All of the testing was performed before and after the intervention period. Test procedures were identical on both occasions and performed by the same examiner.

#### 3.2 Participants

Selection for this study was done by randomisation. Advertisements were placed in local newspapers as well as on local radio. Referrals by general practitioners were also used. Potential subjects were then contacted by telephone and sent all of the required paperwork by e-mail or fax. Inclusion and exclusion criteria were as follows:

- Inclusion criteria
  - Both male and female subjects
  - Between the ages of 20-55
  - Suffering from back pain for at least 12 weeks
  - No neurological symptoms
  - With or without radiating symptoms in the legs (included as long as there were no neurological symptoms)
  - Score of at least 35 of the visual analogue scale for pain
  
- Exclusion criteria
  - Previous spinal surgery
  - Spinal pathology and discogenic disease
  - Any 'red flag' symptoms
  - Current pregnancy
  - On-going disability and injury compensation cases

- Exercise therapy modality treatment within the last six months
- Body mass index (BMI kg/m<sup>2</sup>) of over 40 (severely overweight)

All subjects had to complete a screening questionnaire to identify any potential 'red flag' diseases. The questionnaires were then screened by a medical specialist (rheumatologist) to try and identify any possible warning signs. Subjects were given a copy of the questionnaire to read in advance, and asked to sign the document and state that they understood all of the risks and rewards involved in the study.

All subjects also had to complete and sign an informed consent form that was approved by the University of Pretoria's Faculty of Humanities as well as the Faculty of Health Science.

All of the testing procedures as well as the 12 week intervention programme were explained in detail to the subjects during the first meeting.

A random table of numbers was used, as found in Thomas and Nelson (2001). Each subject was allocated a number, which also assured anonymity when the data was analysed. They were then randomly allocated to either the conservative exercise group or the progressive-aggressive exercise group.

### **3.2.1 History of the Subjects**

In total 45 subjects were recruited for the study and randomly assigned to either the conservative exercise group (n = 20) or the progressive-aggressive exercise group (n = 25). However, 13 dropped out of the study before they were screened. Of the 13 drop-out figure, one subject's symptoms were too severe due to an advanced spondyloarthritis, three lived too far away and could not undertake the regular journey, one subject's spouse fell severely ill, one subject had to go back to her home country, one fell down a flight of stairs and was recommended by the medical doctor to not do any exercise for at least six months and the rest (six subjects) dropped out due to work commitments/problems.

Of the subjects that remained 32 were screened and started on the exercise programmes. During the course of the study six subjects dropped out of the study

before they had completed four weeks (three had too many work commitments, one had too many family commitments, one emigrated and one lost interest). Four subjects dropped out after completing four weeks, but not reaching eight weeks (one emigrated, one had transport problems, two had work problems). Another subject dropped out after completing eight weeks but not getting to 12 weeks due to transport problems, while 21 subjects completed the full 12 week intervention programme (n = 10 in the conservative exercise group and 11 in the progressive-aggressive group). Only the subjects who were screened will have their data used in the study.

### **3.2.1.1 The Use of Low Numbers in the Present Study**

The use of low numbers in similar studies is not uncommon. Subjects with low back pain are difficult to recruit for studies involving exercise therapies. This may be because of a number of factors. Greater numbers are not always possible if subjects are either not inpatient-based or outpatient-based.

During a review of the literature by Moreau *et al.* (2001) concerning the testing of the low back extensor muscles it was reported that studies using 10 subjects or less showed some of the best reliability results, although some would argue that the sample size is too small to draw any conclusions from it.

Cleland *et al.* (2005) reported that a sample size of 15 subjects per group provides greater than 80% power to detect both statistically significant and clinically meaningful differences between groups. The researchers from that study screened 117 subjects and 81 subjects (69%) did not satisfy the inclusion and exclusion criteria for participation. Six (5%) refused participation, which, as they stated, was due to very strict inclusion and exclusion criteria (Cleland *et al.*, 2005). They only used 30 participants in their study. It is not mentioned how many subjects completed the study and it is thus assumed that 30 completed the study.

Radebold *et al.* (2000) used 17 healthy subjects and 17 subjects with chronic low back pain in a study to determine if subjects with chronic low back pain reacted differently to healthy subjects when a sudden load is released. They hypothesised that delayed muscle response and altered muscle recruitment patterns would

emerge in subjects with chronic low back pain (Radebold *et al.*, 2000). It may be argued that only 17 low back patients participated in their study and there was no timeframe involved; subjects participated in a once-off test.

Similarly, Hodges and Richardson (1996) used 30 subjects for a motor control experiment. Of these subjects 15 were healthy and 15 suffered from chronic low back pain. Their motivation for subject selection was based on strict clinical criteria of chronicity and severity. They argued these to be necessary because of the difficulty in obtaining a homogenous subject group based on current investigative techniques. These techniques are unable to identify a definitive cause for back pain in the majority of subjects (Hodges & Richardson, 1996).

Richardson *et al.* (2002) also used a small population group of healthy subjects for a once-off measurement test. In total they used 13 subjects without a history of low back pain and attempted to gain objective measurement values regarding sacroiliac joint mechanics and its contribution to low back pain management (Richardson *et al.*, 2002).

O'Sullivan *et al.* (2003) used a cross-sectional observational design, which included 15 healthy subjects and 15 subjects with a history of chronic low back pain lasting up to three months. Again the investigators attempted to gather objective data using a small population group.

Also, Moseley *et al.* (2002) used eight subjects and deep muscle electromyographic instruments to measure muscle activation in healthy subjects.

Kankaanpää *et al.* (2005) used 12 healthy subjects and 17 subjects with chronic low back pain to measure muscle fatigue ratios during dynamic exercise. This study used objective data as well as a small sample population group. Their criteria for subject selection were similar to those of the present study. Subjects who had been suffering from low back pain for longer than three months, had not undergone any spinal surgery and suffered from no 'red flag' conditions (nerve root entrapment, spinal cord compression, tumours, osteoporosis, recent spinal fracture,

cardiovascular disease, metabolic disease or acute infections) were used during the study (Kankaanpää *et al.*, 2005).

Linemen *et al.* (2003) used 20 subjects selected for surgery due to disc herniation and measured their muscle repositioning ability. The researchers compared them to healthy controls. Both studies used small sample groups, although the characteristics of the subjects were different in both studies. This shows the difficulty in recruiting subjects with homogenous characteristics for low back pain studies.

Hasegawa *et al.* (2008) used 22 patients in a study to measure lumbar segmental instability with an intraoperative measurement system. This study used a small sample. However, the inclusion criteria were very strict. The study was also very labour intensive, as the subjects were measured by means of radiological imaging as well as being measured surgically.

In many of these studies an experimental group was compared to a control group that consisted of healthy subjects. It has to be noted that both groups used in the present study were homogenous and all suffering from chronic low back pain.

Arokoski *et al.* (2004) used a small population group consisting of a total of nine subjects. Their study involved a 12 week exercise intervention programme comprising four to six exercise sessions per week over the 12 week period, based on an outpatient basis. Their subject population group was very similar to the population group used in the present study. Specific causes of back pain, previous spinal surgery, any 'red flag' condition as well as having suffered from back pain for longer than three months were excluded from the study.

The present study used the same selection criteria for selecting subjects like many of the studies mentioned (Arokoski *et al.*, 2004; Petersen *et al.*, 2002). It also used subjects with chronic low back pain in both the control and experimental groups. Also, the study was very labour intensive, both from the researcher's point of view as well as the subjects' participation. Subjects had to commit to the study for 12 weeks. Work commitments became an issue for many participants. It is because of this that the present study will identify a new term: *Full working capacity adults* (FWCA). All

subjects participating in the study were working full-time, which included anything from 8-12 hours per day. Travel time to work was also considered. The participating subjects were thus neither inpatient-based nor outpatient-based. This concept will be dealt with further in the discussion chapter.

### **3.3 Methods and Materials**

The methods and materials mentioned below were used.

#### **3.3.1 Medical Screening**

All subjects included in the study had to complete a screening questionnaire, which was designed purely to identify potential 'red flag' conditions or anything that could exclude them from the study. After completion the screening questionnaires were reviewed by a practicing rheumatologist and senior lecturer at the University of Pretoria in the department Sport Medicine. Of all those who were screened, none presented with any dangerous symptoms that would exclude them from participating in the study.

#### **3.3.2 Study Design**

The design of the study will be a pre-test/post-test randomised group design. The major advantage of this type of study is that the amount of change produced by the treatment can be measured by measuring the amount of improvement in the experimental group compared to the control group (Thomas & Nelson, 2001). The study was designed to be a pre-test/post-test randomized group design, and has the advantage of being able to compare the control and experimental groups in order to measure the effectiveness of the treatment by observing the amount of change produced by the treatment (Thomas & Nelson, 2001).

In this type of research design subjects are randomly allocated to their respective groups with both groups receiving a pre-test as well as a post-test (Thomas & Nelson, 2001). This type of research has been acknowledged as being the most scientific of all research designs (Thomas & Nelson, 2001). Both the control and experimental groups were measured at pre-test as well as post-test, after they are randomly allocated to their predesigned groups. Acknowledgement has recognized

this type of research design as being one of the most scientific (Thomas & Nelson, 2001).

By default this design threatens internal validity through testing (the effect of one test on subsequent administration of the same test, i.e. a learning effect) but this threat is controlled by comparison between the two groups (Thomas & Nelson, 2001). Age-gender matched controls will also be applied in the design. This type of design can threaten internal validity through a learning effect when the effect of one test on following administration of the same test occurs. However, comparison between the two groups controls this threat. (Thomas & Nelson, 2001). Age-gender matched controls will also be applied in the design.

### **3.3.3 Questionnaires**

Several questionnaires were used in the study. All of the questionnaires were completed by the subjects at pre-test, four weeks, eight weeks and at the end of the study at 12 weeks. All of the selected questionnaires are used extensively in low back pain and physical therapy studies, because they are all valid, reliable, repeatable, sensitive to change and they correlate well with other instruments (Linton *et al.*, 2005; Heymans *et al.*, 2006; Kääpä *et al.*, 2006; Goldby *et al.*, 2006). Questionnaires were selected that would measure self-reported pain, levels of disability, levels of kinesophobia and fear avoidance beliefs.

#### **3.3.3.1 Pain and Disability**

The following questionnaires were used to measure levels of pain and disability:

##### **3.3.3.1.1 The Visual Analogue Scale (VAS) for Pain Measurement**

The VAS consists of a single 100 mm line across the surface of a page. On the left side of the line no pain is indicated, while maximal amount of pain is indicated on the right hand side of the line. Subjects had to indicate how they would rate their own pain by indicating it on the scale (Ostelo & De Vet, 2005).

A score is presented out of a 100 being maximal. The intensity of low back pain is measured to determine the quantitative estimate of how severe the patient perceives their back pain measured by this subjective scale (Kankaanpää *et al.*, 2005; Ostelo



&De Vet, 2005). This instrument has a high test-retest reliability of  $r > 0.95$ , has high criterion related validity with established instruments and is well suited to measure pain intensity (Wewers & Lowe, 1990).

### **3.3.3.1.2 Oswestry Disability Index (ODI)**

The impact of low back pain on daily activities is measured by the Oswestry Disability Index (Fairbank & Davies, 1980). It is used to measure non-malignant spinal disorders and is one of the most common used self-administrated questionnaires (Turk & Marcus, 1994; Doleys *et al.*, 1997; Deyo *et al.*, 1998; Carreon *et al.*, 2008; Mehra *et al.*, 2008). The ODI is also used to measure condition-specific outcomes (Fairbank & Pynsent, 2000).

Low back pain included disability and limitations in daily tasks and leisure time activity is included in a 10 section questionnaire (Fairbank & Davies, 1980; Ostelo & De Vet, 2005; Mehra *et al.*, 2008). Each section has a score of 0-5 with 0 representing no disability and 5 representing maximal disability (Ostelo & De Vet, 2005; Mehra *et al.*, 2008).

Totals for the questionnaire is determined by means of a percentage score, where the index is calculated by dividing the summed scores by the total possible score, and is then multiplied by 100 (Ostelo & De Vet, 2005; Mehra *et al.*, 2008). The total score is reduced by 5 for every question that is not answered, and the highest scoring statement is recorded when more than one answer is marked (Mehra *et al.*, 2008).

Mehra *et al.* (2008) reported that the question frequently not answered related to the subject's sex life and this result was also found in the present study. The Oswestry Disability Index has been found to be reliable, valid and sensitive to change (Fisher & Johnston, 1997).

### **3.3.3.1.3 Functional Rating Index (FRI)**

According to Feise & Menke (2001) the Functional Rating Index is an instrument purposely designed to quantitatively measure the subjective perception of function and pain of the spinal musculoskeletal system in a clinical setting. In particular, it

evaluates the patient's subjective report of his/her ability to perform dynamic movements of the neck and back and/or withstand static postures.

It was developed to provide an assessment instrument that has clinical value (i.e., easy and fast for both the patient and the health care team) yet quantifies the patient's current state of pain and dysfunction in a reliable and valid manner for spinal conditions (Feise & Menke, 2001).

According to Feise and Menke (2001) the FRI instrument contains 10 items that assess both pain and function of the spine and its musculoskeletal system. Of these 10 items, 8 refer to activities of daily living that might be adversely affected by a spinal condition, and 2 refer to two different attributes of pain. The use of both pain and the loss of function in spinal conditions are better to use in combination, since many spinal conditions contain a combination of the two factors.

According to Feise and Menke (2001) using a 5-point scale for each item, the patient ranks his or her perceived ability to perform a specific task and/or the quantity of pain at the present time ("right now, at this very moment") by selecting one of the five response points that are anchored by polarized statements (0 = no pain or full ability to function; 4 = worst possible pain and/or unable to perform this function at all).

For scoring purposes, the 10 items of the FRI were totalled according to the responses given, divided by the total possible points available and then multiplied by 100 to produce a percentage value, as recommended by Feise and Menke (2001). The range of possible scores is zero percent (no disability) to 100 percent (severe disability). The higher the score, the higher the perceived pain and dysfunction (Feise & Menke, 2001).

### **3.3.3.2 Fear Avoidance**

The following questionnaire was used to evaluate fear avoidance:

#### **3.3.3.2.1 Fear Avoidance Beliefs Questionnaire (FABQ)**

The FABQ is an instrument that contains 16 items and is divided into two subscales. The first is a 4-item subscale regarding physical activities and the fear avoidance

beliefs towards them (FABQ/pa). The second is a 7-item subscale regarding work and related activities and the fear avoidance beliefs towards them (FABQ/w) (Swinkels-Meewisse *et al.*, 2003).

Items are scored on a 7-point Likert scale ranging from 0 (strongly disagree) to 6 (strongly agree). Total score for the FABQ/pa ranges from 0-24 and the total score for the FABQ/w subscale ranges from 0-42 (Swinkels-Meewisse *et al.*, 2003). The two subscales show sound internal consistency (Swinkels-Meewisse *et al.*, 2003).

### **3.3.3.3 Kinesiophobia**

The Tampa scale for kinesiophobia (TSK) was used to measure fear of movement.

#### **3.3.3.3.1 The Tampa Scale of Kinesiophobia**

A fear of movement and activity has been suggested to be measurable by the Tampa Scale of Kinesiophobia (Kori *et al.*, 1990; Vlaeyen *et al.*, 1995). This instrument consists of a questionnaire that includes a 17-item set of questions and was developed as a means of identification of a fear of injury because of movement and/or activities (Swinkels-Meewisse *et al.*, 2003).

Items are scored on a 4-point Likert scale with scoring possibilities ranging from 'strongly disagree' (score = 1) to 'strongly agree' (score = 4) (Swinkels-Meewisse *et al.*, 2003). The scores of items 4, 8, 12 and 16 were reversed and then calculated.

### **3.3.3.4 Exercise Intensity**

This study attempted to measure the intensity of the exercises as well as the exercise programmes.

#### **3.3.3.4.1 The Borg Rate of Perceived Exertion (RPE) Scale**

For the purpose of this study the Borg 6 to 20 rating of perceived exertion (RPE) scale was used in order to determine the intensity of different exercises in different programmes and to determine whether the intensity of the exercise was too easy or too difficult and whether the change from one programme to the next was sufficient.

To induce a training effect but not to influence exercise compliance in a harmful way or aggravating symptoms is a challenge of remedial exercise therapy that needs to be investigated (Dawes *et al.*, 2005). The monitoring of exercise intensity during exercise in healthy subjects has been measured effectively by the rate of perceived exertion (RPE) scale (Borg *et al.*, 1987). Clinical populations have also been monitored using this scale when exhibiting symptoms (Bateman *et al.*, 2001; Barker *et al.*, 2003).

Below is an example of the RPE scale used in the present study.

**Table 3.1: Borg RPE Scale (Williams & Eston, 1996)**

6 No exertion at all	14
7 Extremely light	15 Hard (Heavy)
8	16
9 Very light	17 Very hard
10	18
11 Light	19 Extremely hard
12	20 Maximal exertion
13 Somewhat hard	

An ability to sense effort has been reported to be well developed and in regular use in humans (Williams & Eston, 1996). Humans can sense when to stop or to continue during vigorous physical activity and can account overall feelings of exertion to particular sites, such as in the chest or arms and whether a sensation becomes maximal (Williams & Eston, 1996; Dawes *et al.*, 2005).

Humans can numerically scale various levels of exercise to which they are subjected to with some experience of physical activity (Williams & Eston, 1996).

As exercise intensity increases, there is a linear increase in the rate of perceived exertion in the 6 to 20 RPE scale, which is closely linked with physiological responses such as heart rate and oxygen use, which also increases linearly (Williams & Eston, 1996). Exertional symptoms such as breathlessness and muscle pain also increase accordingly (Borg *et al.*, 1985; Borg *et al.*, 1987).

As the subjective perception of the exercise changes, the RPE consists of numbers that are anchored to verbal responses that will change as the subject experience subjective changes (Williams & Eston, 1996). Subjects had to rate their own perception of a specific exercise with a number value on the scale for each exercise and this was then compared afterwards.

### **3.3.4 Physical Testing**

The following tests were used to assess physical status:

#### **3.3.4.1 Neurodynamic Testing**

Popular accessory testing in the investigating of musculoskeletal injuries such as the straight leg raise test and the slump test have recently emerged and is used in the assessment of neural tissue mobility and sensitivity to mechanical stress (Herrington *et al.*, 2007). The categorizing of patients into groups with dissimilar prognosis and measured disease severity is the goal of diagnostic instruments in low back pain cases (Mens *et al.*, 2001). The value of both physical examinations and radiographic measurements is limited however (Mens *et al.*, 2001). A need thus exists for the use of simple tests with high validity, sensitivity and specificity (McCombe *et al.*, 1989).

##### **3.3.4.1.1 Straight Leg Raise Test**

Used as an aid in the diagnosis of low back pain conditions, the passive straight leg raise test is frequently used to assist clinicians (Jönsson & Strömqvist, 1995; Jönsson & Strömqvist, 1996). There is however, a lot of doubt in the best use of the test in terms of how it should be performed, the mechanism of its limitation and the clinical significance (Van den Hoogen *et al.*, 1996).

Pain during the passive straight leg raise test has been suggested to be because of the compression of the nerve root (O'Connell, 1943; Falconer *et al.*, 1948). This has

been suggested to be caused by the sciatic nerve root being unable to move away from a disc protrusion, since it is fixed between the dura and the intervertebral foramen and thus the ensuing compression and induced traction-generated mechanism can cause pain (Inman & Saunders, 1942; Falconer *et al.*, 1948). The path of the nerve root movement has been reported as caudal but also as lateral towards the pedicle and so towards any posterolateral disc herniation (Rebain *et al.*, 2002).

The dura might be a contributor in the production of pain since it has been reported that the dura moves less than the intrathecal nerve root at the pedicle and thus experiences more strain (Rebain *et al.*, 2002).

It has been suggested that an effect on the outcome of the passive straight leg raise test can be influenced by a disc protrusion (Rebain *et al.*, 2002). Low back pain can result during a central prolapsed of the disc (Falconer *et al.*, 1948); both leg pain and back pain can result from a posterolateral protrusion (Rebain *et al.*, 2002) and leg pain alone can be produced by a lateral protrusion (Edgar & Park, 1974). It has also been reported that an improvement in the passive straight leg test result might not occur even if a decline in the size of the protrusion over time occurs (Thelander *et al.*, 1992).

Examination of the exit of the sciatic nerve from the pelvis during the straight leg raise test occurs only after 2.54-5.08 cm of leg raising and is evident after 20-30 degrees at the intervertebral foramen (Rebain *et al.*, 2002). The greatest amount of motion takes place at the L5-S2 level at 60-80 degrees of the passive straight leg raise test, but little movement occurs at L3 and higher (Inman & Saunders, 1942; Goddard & Reid, 1965). Movements of 4-5 mm at the S1 nerve root and 3 mm at the L5 nerve root has been reported (Inman & Saunders, 1942; Goddard & Reid, 1965). However, there is a decline of movement reported with age, probably due to adhesion from the sciatic nerve and the neighboring tissue (Goddard & Reid, 1965).

Damage to related ligamentous structures and collateral creation of an inflammatory focus over the dural cuff of the nerve have also been reported as possible pain producing mechanisms during the passive straight leg raise test (Inman & Saunders,

1942). Other factors could also include possible nerve root edema (Pennybacker, 1940; Holmes & Sworn, 1945; Falconer *et al.*, 1948); nerve root irritation (Goddard & Reid, 1965; Epstein *et al.*, 1972) and intervertebral foramen venous obstruction (Hoyland *et al.*, 1989; Kobayashi *et al.*, 1993).

The passive straight leg raise test has been reported to be a test for the assessment of neural tension and hamstring length (Loudon *et al.*, 1998). A defensive hamstring muscle reaction can lead to a restriction in the result of the passive straight leg raise test in order to protect the structure from possible damage (Goddard & Reid, 1965; Goeken & Hof, 1991; Ismaiel & Porter, 1992; Goeken & Hof, 1993; Hall *et al.*, 1995; Hall *et al.*, 1998).

A limited extensibility of the hamstring muscles in asymptomatic subjects and restricted extensibility produced by a defensive reaction to avoid nerve stretch is not able to be distinguishable by the passive straight leg raise test (Goeken & Hof, 1991; Goeken & Hof, 1993; Goeken & Hof, 1994). Hall *et al.* (1995) and Hall *et al.* (1998) supported these conclusions. They further reported that radiculopathy patients showed hamstring muscle response before reporting onset of pain. Hall *et al.* (1998) reported that such hamstring defense reaction in protecting inflamed nerve roots reflected a heightened mechanosensitivity of the nervous system. It is thus clear that the passive straight leg raise has to be interpreted with caution.

For the purpose of this study, the technique described by Loudon *et al.* (1998) will be used, in which the subject was placed supine on an examining table with the arms at the side. The subject's leg was gradually lifted into hip flexion while keeping the knee extended. Adding passive cervical flexion, dorisiflexion and plantar flexion may add tension to several nerve pathways by adding sensitization. This was added after the subject's leg reached maximum length. Reproduction of back or leg symptoms for tightness indicated a positive finding. Both legs were tested in this way. The same examiner performed the tests each time.

If the subject experienced leg symptoms when lifting the unaffected leg, it places tension on the nerve root on the unaffected side together with causing tension centrally along the midline of the cauda equina and to the nerve roots on the

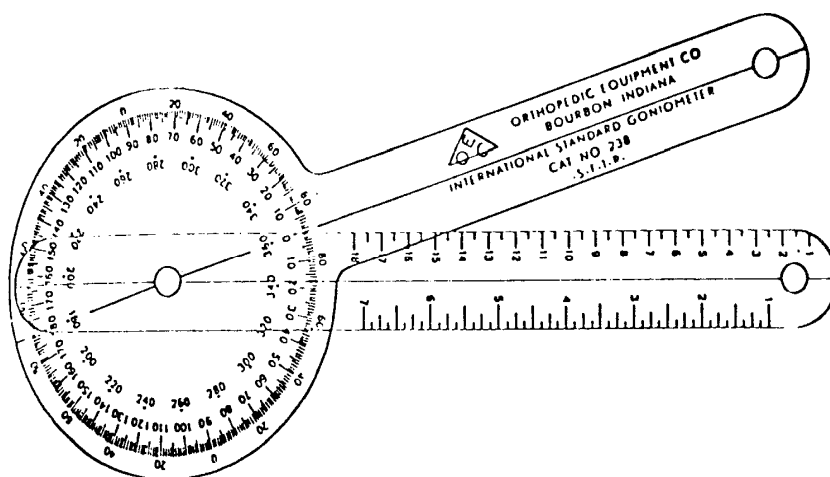


opposite leg (McGill, 2002). Simultaneous cervical flexion can also sometimes produce pain. An organic sign of disc lesion is when pain is reproduced on the symptomatic side, which may be a more central lesion (McGill, 2002). Neural tension can be indicated when pain is produced along specific pathways (Loudon *et al.*, 1998).



**Figure 3.1 : The Straight Leg Raise Test**

A protractor goniometer was used to measure the total amount of hip flexion after the subject's leg reached maximum height (Borms & Van Roy, 1996).



**Figure 3.2: The International Standard Protractor Goniometer**

The angle between two bony landmarks is measured by the goniometer, and the angle is recorded when the maximum height is reached. A pelvic tilt occurs with a consequent reduction of the lumbar lordosis during straight leg raise testing (Borms & Van Roy, 1996). Because of this, the straight leg was raised to its maximum level

of perceived comfort. When the subject experienced discomfort, either pain or stiffness in the leg or back, the amplitude measurement was recorded along with reproduction of symptoms. The landmarks used for this test were the tip of the greater trochanter and the lateral femoral epicondyle, according to the recommendations of Borms & Van Roy (1996).

However, it has to be noted that, as reported by Herrington *et al.* (2007), even asymptomatic subjects tend to have positive neurogenic response to structural differential of the straight leg raise test and the slump test. The finding of positive structural differentiation does not necessarily imply the presence of neural pathology.

Neural pathology is thus not necessarily indicated by the finding of positive structural differentiation. To be regarded as being positive test result of an underlying pathology, the test outcome has to be greater than for asymmetric subjects found by studies such as those conducted by Herrington *et al.* (2007). It also shows a great difference in symmetry between limbs (Herrington *et al.*, 2007). This consideration has to be taken into account to avoid possible false-positive results and this is also why other tests have been considered in the present study.

#### **3.3.4.1.2 The Slump Test**

Neural tension is very often measured by the Slump Test (Loudon *et al.*, 1998). Tensing of the sciatic nerve and irritation of the lumbar nerve roots is the goal of this specific test (McGill, 2002).

During this test, the subject is seated on an examination table and is put through a series of motions, stopping if any symptoms occur or if any resistance to motion is occurred. The subject started by placing their hands behind the back, rounding the shoulders and flexing the neck, after which the examiner used one hand to passively extend one of the subject's legs (Loudon *et al.*, 1998). The sciatic nerve is not the cause of pain if pain levels were not increased during the flexion of the neck (McGill, 2002).

The test is performed by first dorsiflexing the foot, and early resistance or a reproduction of back and/or leg symptoms indicate a positive finding. When lifting the head causes a release of pressure, it confirms the existence of neural tension. The test was performed on both sides (Loudon *et al.*, 1998). The same examiner performed the test on all occasions.



**Figure 3.3 : The Slump Test**

#### **3.3.4.2 Muscle Endurance Testing**

Biering-Sorensen (1984) suggested that those who are at greater risk for future back problems show a noticeable decrease in torso muscle endurance. McGill (2002) however, suggested that a better identification of those who have suffered from low back problems in the past can be found in the balance of endurance among the flexor, extensor and lateral musculature of the torso. Because all these muscle groups are involved in spine stability during practically any task, the endurance of all three muscle groups should be measured.

Simple tests that isolate these muscle groups should be used, and therefore the following tests have been selected because they all have a high reliability coefficient and are relatively easy to perform (McGill *et al.*, 1999):

##### **3.3.4.2.1 The Sorenson Back Extension Test (The Ito Test Version)**

The extensor muscle group of the trunk is measured very effectively by this test, especially the paraspinal muscles are very successfully measured, which includes the multifidus muscle (Ng *et al.*, 1997; Arokoski *et al.*, 1999) as well as the hip extensor muscle group (Demoulin *et al.*, 2004).

In those with chronic low back pain, it has been found that these subjects have a significantly decreased position holding time for this test (Hansen, 1964; Biering-Sorensen, 1984; Salminen *et al.*, 1992; Hultman *et al.*, 1993; Jorgensen, 1997; Simmonds *et al.*, 1998; Latimer *et al.*, 1999; Novy *et al.*, 1999;). Thus, a decline in isometric endurance of the trunk extensor muscles has been argued to be associated with chronic low back pain and its effects (Demoulin *et al.*, 2004).

Greater levels of severity among those with chronic low back pain have been suggested to be associated with poorer test performance during extensor endurance tests (Mannion & Dolan, 1994; Lindstrom *et al.*, 1995; Jorgensen, 1997). This makes the Sorensen test very applicable to use with subjects with chronic low back pain.

McGill (2002:226) reported that: “...*the back extensors are tested in the ‘Biering-Sorensen position’ with the upper body cantilevered out over the end of a test bench and with the pelvis, knees and hips secured. The upper limbs are held across the chest with the hands resting on the opposite shoulders. Failure occurs when the upper body drops from the horizontal position.*”

The test is stopped after four minutes in those subjects who experience no issues with the test (Demoulin *et al.*, 2004). Biering-Sorensen (1984) reported that during a one year period, a position-holding time of less than three minutes in males can prompt low back pain, whereas a holding time of more than 3.3 minutes reports a low percentage of low back pain during a one-year period. A risk of low back pain with a holding time of less than 58 seconds has been predicted to increase the risk three-fold (Luoto *et al.*, 1995). In healthy subjects, the mean extensor endurance times for mixed gender groups range from 77.76 – 129 seconds (Mannion & Dolan, 1994; Luoto *et al.*, 1995; Moreland *et al.*, 1997; Simmonds *et al.*, 1998).

Healthy women on average typically have a tendency to produce longer extension endurance times than healthy men, with women averaging 142 – 220.4 seconds while men scores an average of 84 – 195 seconds (Mannion & Dolan, 1994; Mannion *et al.*, 1997; Kankaanpää *et al.*, 1998).

Mixed gender groups with low back pain scores a mean endurance time between 39.55 – 54.5 seconds, while men with low back pain scores 80 - 194 seconds and women score 146 – 227 seconds (Moffroid *et al.*, 1994; Simmonds *et al.*, 1998; Moreau *et al.*, 2001). For low back pain cases in men and women, the Sorensen test has been shown to have prognostic worth (Adams *et al.*, 1999; Sjollic & Ljunggren, 2001).



**Figure 3.4: The Sorensen Back Extension Test**

For the purpose of this study, the test was modified according to Ito *et al.* (1996) with two differences being the subject's feet being held down and the other that instead of hanging over the edge of a table, the subjects were placed in a prone position on an exercise mat on the floor. Arms were placed alongside the body and the feet were held in place. Because arm position influences the location of the centre of gravity, the modification made with the arms will affect the mass moment of the upper body and therefore influence the test performance (Mayer *et al.*, 1999).

Upon starting, the subjects would lift their upper bodies from the ground until their chests were slightly off the floor while flexing the neck as much as possible without creating discomfort in the neck. This in turn increased the activity level of the erector spinae muscles (Ito *et al.*, 1996). The position was then held for as long as comfortably possible. Subjects were allowed to stop when experiencing discomfort, either in the form of pain or muscle fatigue.



**Figure 3.5 : The Ito Test**

Some subject experience increased levels of difficulty with this test, no matter how much contraction takes place (Moreau *et al.*, 2001). Biering-Sorensen (1984) reported that 24% of their sample could not complete the test due to back pain followed by pain in the legs or abdomen. Latikka *et al.* (1995) reported a 50% failure rate because of back pain or fatigue. Other side-effects that have been recognized with the test include cramps of the calves, neck pain, discomfort in the head, abdominal pain and breathlessness (Latikka *et al.*, 1995; Moreland *et al.*, 1997).

The time the position was held, was measured with a stopwatch. The reason for stopping was also noted. Test results of subjects who stopped for pain reasons might not have been an accurate reflexion of muscle performance (Biering-Sorensen, 1984; Mannion & Dolan, 1994; Latikka *et al.*, 1995; Moreland *et al.*, 1997; Latimer *et al.*, 1999). It is thus very important to note the reason for stopping and to be able to compare from pre-test to post-test.

It has been suggested that the Ito test might result in less spinal loading than the Sorensen test, which was why it was selected in the present study (Demoulin *et al.*, 2004). The input of the hip extensor muscles has been suggested to be smaller because the lower body is not fixed into position with straps (Plamondon *et al.*, 2002).

Lasting pain and adverse effects with the Sorensen test have seldom been reported, and is thus a relatively safe submaximal test and can be used safely and successfully (Simmonds *et al.*, 1998). This is because the extensor muscles of the

low back contract well below the threshold of the maximal voluntary contraction (MVC) (Moffroid *et al.*, 1993; Mayer *et al.*, 1995).

Subjects with no low back pain sustained the endurance contraction at the following levels:

- Slim, strong subjects: 20-25% of MVC (Jorgensen & Nicolaisen, 1986)
- Subjects with no low back pain or low back pain that does not prevent work: approximately 60% of MVC (Hultman *et al.*, 1993)
- Untrained and overweight subjects: approximately 70-75% of MVC
- Subjects with chronic low back pain: approximately 85% of MVC (Jorgensen & Nicolaisen, 1986)

Even though the Ito test is a good variant, the Sorensen test is more recommended than the Ito test (Demoulin *et al.*, 2004). This is because the Ito test version still requires more validation before it can be considered a valid instrument (Ito *et al.*, 1996). The test is agreed upon to be very cost effective and easy to perform, can be done in a short time and does not necessitate any special equipment, either in its original form or some sensible variation of the test (Moreau *et al.*, 2001). Subject motivation unfortunately plays a big role in the performance of the test, and low levels of motivation due to factors such as fear avoidance behaviour can influence the outcome of the test (Kankaanpää *et al.*, 1998).

#### **3.3.4.2.2 Side Bridging Endurance Test**

This test measures the lateral muscle group. The test is described in McGill (2002: 225): “*The lateral musculature is tested with the person lying in the full side-bridge position. Legs are extended, and the top foot is placed in front of the lower foot for support. Subjects support themselves on one elbow and on their feet while lifting their hips off the floor to create a straight line over their body length. The uninvolved arm is held across the chest with the hand placed on the opposite shoulder. Failure occurs when the person loses the straight-back posture and the hip returns to the ground.*” The only change made to the test used in the present study was that the subjects were required to place the uninvolved hand on the hip, as shown in fig. 3.6.





**Figure 3.6 : The Side Bridging Endurance Test**

#### **3.3.4.2.3 Flexor Endurance Test**

This test measures the flexor muscle group. The test was performed with the subject in a sit-up posture with the back forming a 60° angle with regard to the legs. This angle was achieved with the subject placing the hands on the knees for support and leaning back until the desired angle was achieved. The test was started when the subject let go of the knees and placed them on the shoulders. Both knees were flexed 90°, the arms folded across the chest with the hands placed on the opposite shoulder. The feet were kept in place by another person holding them down. To begin, the subject held the isometric posture as long as possible. Time was measured using a stopwatch. Failure was determined to occur when the subject could no longer continue. Subjects were also allowed to stop if they felt pain or muscular fatigue in their low back (McGill, 2002).



**Figure 3.7: The Flexor Endurance Test**

### 3.3.5 The Exercise Programmes


The intervention consisted of two separate programmes. The first programme was for the control group. This group received an exercise programme that was considered to be conservative in nature. They completed the programme twice per week with a session lasting for approximately 35-40 minutes. This programme remained unchanged throughout the 12-week intervention timeframe.





The second programme was considered more aggressive in terms of the exercises performed as well as the intensity of the programmes. The subjects completed the programme for four weeks after which it was progressed to a more difficult level. After another four weeks (eight weeks in total) the programme was progressed again to a more difficult level and was again completed for four weeks (12 weeks in total). This group was the experimental group. The programme was also completed twice per week with a session lasting for approximately 60 minutes, along with the back school session.

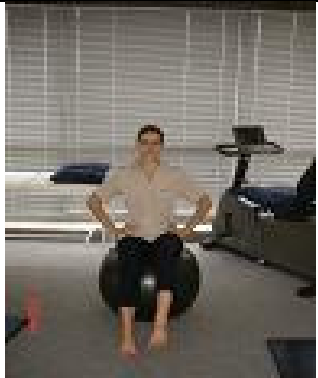



All of the exercises in the experimental group were performed with stabilisation (abdominal bracing). All of the exercise sessions were supervised by the principle researcher who is a qualified rehabilitation specialist. Below follows the complete descriptions of the exercise programmes.


#### 3.3.5.1 Control Group (Conservative Exercise Programme)

Resting time between sets was 20 seconds.

Illustration	Exercise	Sets	Reps
	<b>Cycling:</b> This was performed on a recumbent cycle, model Vision Fitness R2150. Subjects cycled for five minutes at Level 2 (43-55 watt) at an RPM (revolutions per minute) of 60-70.	5 min.	

	<p><b>Both Knees to Chest Stretch:</b> Performed with the subject in supine position. Subject started by pulling up both knees towards the chest, lifted to the position of mild discomfort, held for 12 seconds (Prentice, 2004).</p>	2	12 sec.
	<p><b>Hamstring Stretch:</b> Performed with subject in supine position. The subject lifted up one leg, placed hands at the back of the knee, pulling the leg up with knee slightly bent, stretching the hamstring. The leg was lifted to a position of mild discomfort. The opposite leg was placed flat on the ground. The position was held for 12 seconds.</p>	2 sets each leg	12 sec.
	<p><b>Periformis Stretch:</b> Performed with subject in supine position. Ankle of leg was placed on knee of opposite leg, hands behind the knee. The knee was pulled towards the chest (Prentice, 2004). Held position for 12 seconds.</p>	2 sets each leg	12 sec.
	<p><b>Roll Both Knees to Side:</b> Performed with subject in supine position. Arms were placed outstretched to assist with stability. Knees were bent and placed together. Both feet were lifted off the ground about 10 cm. Knees were then kept together and rolled from side to side, slowly and with control, only up to the point of comfort (Prentice, 2004).</p>	2	10 to each side

	<p><b>Sit on Stability Ball:</b> Performed with subject sitting on a 75 cm stability ball. Hands were placed on hips. Subjects were then asked to lift one leg at a time about 5 cm off the ground, balance in the position for a couple of seconds and repeat with the other leg. Subject had to keep upright without counterbalancing due to altered stability position.</p>	3	30 sec.
	<p><b>Alt Superman on All-fours:</b> Subject started in the all-fours position with the hands under the shoulders and the knees under the hips. The opposite arm and leg were raised simultaneously and only up to horizontal level. The position was then held for 5 seconds. Arm and leg then returned to starting position and the other opposites were raised and held. Subject maintained neutral spine (McGill, 2002).</p>	2	4 each side
	<p><b>Hip Lifts (Feet Flat on Floor):</b> Subject started in the supine position with knees bent and feet flat on the floor. The arms were kept next to the sides. The hips were then lifted until they were fully extended. The position was held for 5 seconds. The hips were lowered and the exercise repeated (Prentice, 2004).</p>	2	10
	<p><b>Prone Alt Leg Lifts:</b> Subject started in the prone position with a pillow under the abdomen to help maintain neutral spine. One leg was lifted until the foot was about 10 cm off the ground with the</p>	2	6 each leg





	leg kept straight. The position was held for 5 seconds. The leg was lowered and exercise repeated with the other leg (Prentice, 2004).		
	<p><b>Prone Alt Arm and Leg Lifts:</b> Subject started in the prone position with a pillow under the abdomen to help maintain neutral spine. The opposite arm and leg were lifted simultaneously about 10 cm off the ground. Both the arm and the leg had to be kept straight. The position was held for 5 seconds. The arm and the leg were lowered and repeated on the other side (Prentice, 2004).</p>	2	6 each side

### 3.3.5.2 Experimental Group (Progressive-Aggressive Programme)





This programme was divided into three progressive exercise programmes. Selected exercises were made to be more difficult from one programme to the next. This was done to increase the intensity of each programme. The exercises were also more aggressive and thus harder to perform than the control group exercises. Each programme was performed for four weeks (eight sessions) before it was progressed to the next programme. Resting time between sets was 30 seconds.

#### 3.3.5.2.1 Programme 1


This programme was performed from the start of the programme to the end of Week 4.

Illustration	Exercise	Sets	Reps
	<p><b>Cycling:</b> This was performed on a recumbent cycle, model Vision Fitness R2150. Subjects cycled for 5 minutes at Level 2 (43-55 watt) at a RPM (revolutions per minute) of 60-70.</p>	5min	
	<p><b>Hamstring Stretch with Foot Flexion:</b> Performed with subject in supine position. Lifted up one leg, placed hands at the back of the knee, pulled leg up with knee slightly bent until the hamstring was stretched. Subject then performed 20 plantar/ dorsiflexion step-off movements with the foot. Opposite leg was placed flat on the floor.</p>	3 each leg	20
	<p><b>Side Lying Quadricep Stretch:</b> Subject lay on her side. The top leg was bent and the foot grasped with the hand. The heel of the foot was pulled towards the buttocks to stretch the quadricep muscle. Position was held for 12 seconds.</p>	3 each leg	12 sec.
	<p><b>Lat Pulldown to the Front:</b> Subject was seated in a standard lat pulldown machine. The bar was grasped with both hands slightly wider than shoulder width. The bar was pulled down towards the chest and in front of the face. This enhanced the role of several spinal extensors, particularly the latissimus dorsi (McGill, 2002). Weight selection: men = 3 plates (12 kg); women = 2</p>	3	15




	plates (7 kg).		
	<b>Side Bridging (on Knees):</b> Subject lay on her side with the knees bent 90°, supported on the elbow and hip. The free hand was placed on the hip. The torso was then straightened until the body was supported on the elbow and the knee. Held position for 15 seconds (McGill, 2002).	3 each side	15 sec.
	<b>High Cable Horizontal Adduction (Downwards):</b> Subject stood in a cable pulley machine and gripped the handle with one hand in an extended abducted position. The arm was kept straight throughout the movement. The arm was then adducted towards the midline of the body and in line with the navel, and then slowly released back to the starting position. Torsion forces had to be resisted by keeping the body straight.	3 each arm	15
	<b>Hip Lifts with Feet on Bench:</b> Subject started in the supine position with the feet on a 46 cm bench in a 90° angle with the arms next to the sides. The hips were raised off the floor until the hips were in full extension. The hips were then slowly lowered and the exercise was repeated.	3	15
	<b>Alt Superman on Stability Ball:</b> Subject started in a prone position with a 75 cm stability ball under the abdomen, with hands and feet placed on the ground. The alternative arm and	3	6 each side (12 total)






	<p>leg were raised until horizontal. The position was held for 5 seconds. Both limbs were slowly lowered until on the ground again. The other pair of opposites was then raised. This had to be done while maintaining balance on the ball.</p>		
	<p><b>Abdominal Crunches (Feet on Bench):</b> Subject started in the supine position with the feet on a 46 cm bench at a 90° angle with the hands behind the head. Eyes had to be kept on the ceiling throughout the entire exercise. The shoulder blades were then raised off the floor, with hands supporting the head and neck. The body was then lowered and the movement repeated.</p>	3	20




### 3.3.5.2.2 Programme 2

Exercises from the first programme are now progressed to increase their difficulty level. It was performed from Week 4 to Week 8. Exercises as well as progression techniques will be discussed.


Illustration	Exercise	Sets	Reps
	<p><b>Cycling:</b> Intensity was increased as follows: Level was increased to 3 (65-75 watt) and the RPM was increased to 65-75.</p>	5 min.	

	<p><b>Hamstring Stretch with Step-off:</b> Subject started in the supine position. Leg was held up with rope or towel, stretched for 12 seconds. The subject performed 12 plantar/dorsiflexion step-offs with leg in extended position. After the 12 step-offs the leg was pulled slightly further back and held for another 12 seconds. The non-involved leg lay flat on the ground.</p>	<p>3 each leg</p>	<p>12;12; 12</p>
	<p><b>Side Lying Quadriceps Stretch:</b> Stayed the same as in the first programme.</p>	<p>3 each leg</p>	<p>12 sec.</p>

	<p><b>Lat Pulldown to Front:</b> Subject was seated in a standard lat pulldown machine. The bar was grasped with both hands slightly wider than shoulder width. The bar was then pulled down towards the chest and in front of the face. This enhanced the role of several spinal extensors, particularly the latissimus dorsi (McGill, 2002). The intensity of this version of the exercise was increased by adding more repetitions. Subjects now performed 25 repetitions. The weight stayed the same.</p>	<p>3</p>	<p>25</p>
---	---	----------	-----------


	<p><b>One arm DB Row:</b> Subject stood with the same arm and same leg placed on a 46 cm bench. The other leg was placed on the floor to give a wide balance position. The other arm held a hand-weight. The weight was raised to the iliac crest with the elbow raised towards the ceiling. Torsion was resisted by bracing the abdominal muscles. The weight was then lowered and the movement repeated. The upper back had to be kept straight and parallel to the floor (Delavier, 2001). The following weight selection was used: men → 5 kg, women → 2 kg.</p>	<p>3 each side</p>	<p>15</p>
	<p><b>Side Bridging (on Feet):</b> Progression of this exercise entailed balancing on the feet and the elbow instead of the knees. The position was still held for 15 seconds.</p>	<p>3 each side</p>	<p>15 sec.</p>
	<p><b>Low Cable Shoulder Flexion (Straight Arm):</b> The subject faced away from a cable pulley machine and gripped a handle in one hand. The shoulder was then flexed to 45° in the sagittal plane. The arm was then returned to the starting position and the movement was repeated.</p>	<p>3 each arm</p>	<p>15</p>



	<p><b>Ball Squat Against Wall:</b> Subject leaned against a wall with a 75 cm stability ball placed in the lower back. Feet were placed forward from the vertical position of the hips, slightly apart. Hands were placed on the hips. The knees were bent to simulate a squat movement. Subject squatted no lower than 45° of knee flexion. Subject then rose back up to the starting position and the movement was repeated.</p>	3	15
	<p><b>Hip Lifts (Feet on Ball):</b> This exercise was performed exactly as in the first programme, except that the feet were placed on a 75 cm stability ball and not on a bench. The subject performed 15 repetitions.</p>	3	15
	<p><b>Alt Superman (Sweeping Hand on Floor Upon Return and Up Again):</b> This exercise was performed exactly as in the first programme, except that instead of alternating the arm and leg combination, the arm and leg just swept the ground upon return and were extended again. No weight was placed back onto that side. One arm and leg combination first finished its repetitions; then the other side was used (McGill, 2002).</p>	3	6 each side (12 in total)





	<p><b>Abdominal Crunches (Feet on Stability Ball):</b> Exactly as in the first programme, except that the feet were placed on a 75 cm stability ball. The repetitions were also increased to 25 per set.</p>	3	25
---	--	---	----

### 3.3.5.2.3 Programme 3




This programme was designed to be the most difficult after progression from the previous two. It was performed from Week 8 to the end of the programme at Week 12. Progression and techniques are discussed below.

Illustration	Exercise	Sets	Reps
	<p><b>Cycling:</b> This exercise was progressed by increasing the level to Level 4 (75-94 watt) and the RPM to 70-80.</p>	5 min.	


	<p><b>Periformis Stretch:</b> Performed with subject in supine position. Ankle of leg was placed on knee of opposite leg, hands placed behind knee. The knee was pulled towards the chest (Prentice, 2004). The exercise was held for 30 seconds.</p>	2	30 sec.
	<p><b>Rotation Stretch:</b> Subject started in the supine position. One leg was bent and placed over the knee of the other leg. The opposite hand in relation to the bent leg was placed on the knee. The bent leg was pulled over to the side to stretch the buttocks. Shoulders had to</p>	2	30 sec.


	be kept down on the ground. Position was held for 30 seconds.		
	<b>Side Lying Quadriceps Stretch:</b> This exercise was performed exactly as in the previous programmes; only it was now held for 30 seconds and not 12 seconds.	2	30 sec.
	<b>Lat Pulldown to Front:</b> This exercise was performed exactly as in the previous programmes. Intensity was increased by means of adding more weight. One plate was added. Men now exercised with 4 plates (15 kg) and women with 3 plates (12 kg). Repetitions were again 15.	3	15
	<b>High Cable Pulldown to Opposite Hip with Both Arms:</b> The subject stood in cable pulley machine and gripped the handle with both hands. The hands were then pulled across the body towards the opposite hip. Controlled torsion forces were encouraged to teach the subject control in the torsional plane.	3 each side	15
	<b>Seated Cable Row:</b> Subject was seated in a standard cable row pulley machine. A V-handle was used. Subject sat upright with feet on the support plates and slightly bent at the knees. The back had to be kept upright during the movement, no flexion or extension was allowed at the hips. The handle	3	15



	<p>was then pulled towards the navel while keeping upright. It was then slowly lowered and the movement was repeated. Men used 2 plates (10 kg) and women used 1 plate (5 kg).</p>		
	<p><b>Ball Squat Against Wall (With Weight):</b> This exercise was performed exactly as in the previous programme, except that the subject held onto a set of hand-weights. Men used 3 kg dumb bells and women used 1.5 kg dumb bells.</p>	3	15
	<p><b>Side Bridging (on Feet, Lifted Side):</b> The starting position was exactly the same as for the previous version of this exercise but was no longer a holding exercise. Instead, the hips were raised in an up and down motion. The subject was instructed to raise the hips towards the ceiling, while keeping the hips extended.</p>	3 each side	12
	<p><b>Hip Lifts With One Leg at a Time (Feet on Bench):</b> The starting position for this exercise is the same as in the previous versions. Intensity is increased by performing the exercise in the same way as previously, but only with one leg at a time. This also increased the volume of the exercise by ensuring that double the amount of sets were done.</p>	3 each leg	10



	<p><b>Alt Superman:</b> The starting position for this exercise was exactly the same as for the other versions. Intensity was increased in the following manner: The arm and leg were held at end range of motion. The subject performed 5 flexion/extension movements with the hand and foot, while the arm and leg were held at the end range of motion.</p>	3	6 each side (12 in total)
---	--	---	---------------------------

	<p><b>Abdominal Crunches (Lying on Ball):</b> This exercise was performed with the same technique as for the other versions in that the hands supported the head and the eyes looked up at the ceiling. Intensity was increased by having the subject lie on a 75 cm stability ball that required more effort to maintain balance. More repetitions were performed. Subjects performed 30 repetitions instead of 25.</p>	3	30
--	--	---	----

### 3.3.6 The Back School

The back school formed the educational part of the rehabilitation programme. An educational document called *The Only Information You will Ever Need to Treat Your Back Pain* was composed from scientific literature and provided information on the following topics:

- **Discussion of correct and proper anatomy**  
These discussions focused on the involved anatomical structures of the lower back and their possible influence in the cause of problems.
- **Discussion of proper ergonomics**

Certain tasks and movements in everyday life may worsen lower back problems. The discussions focused on proper lifting and application techniques.

- **Avoiding bed rest and remaining active with normal activities that were avoided because of back pain**

Bed rest is detrimental to lower back problems. These discussions focused on the importance of avoiding bed rest.

- **Discussion of LBP history**

Previous low back pain injury is a major causal factor for future events. The importance of avoiding risk factors for this reason was discussed.

- **Discussion of risk factor prevention**

Risk factor prevention will drastically decrease the chances of suffering from a future back pain event. Different prevention factors were discussed here.

- **Importance and benefits of exercise**

Exercise therapy is regarded as one of the mainstay treatments for chronic low back pain. These discussions highlighted the importance and benefits of exercise therapy as well as the safety of different exercises.

- **Work to achieve an internal locus of control**

Internal locus of control correlates with a quicker and more complete recovery. These discussions were used to try and facilitate this change of perception in the subjects.

Both groups received an exact same copy of the document to read on their own before the start of the programme. The conservative exercise group only received the document to read. This is referred to as *low-intensity back school*. The experimental exercise group also had to read the document. In addition, they received one-on-one educational sessions discussing all of the topics in the document. This is referred to as *high-intensity back school*.

The educational sessions took place after the training sessions. The educational sessions took between 5-10 minutes each. The topics contained in the back school document were discussed more in-depth with the subjects on an individual basis. This served to provide education and understanding of living with chronic low back

pain and thus provided a large part of the biopsychosocial approach which focuses on education.

## CHAPTER 4

### RESULTS AND DISCUSSION

#### 4.1. Background and Objectives

The objective of the study was to determine the effect of exercise therapy on lower back pain.

#### 4.2. Research Design

A [research] design is used to structure the research, to show how all of the major parts of the research project – the samples or groups, measures, treatments or programmes and methods of assignment – work together to try to address the central research questions. (Research methods, knowledge base; <http://www.socialresearchmethods.net/kb/design.php>).

An experimental design was used in this study. When making use of an experimental design, the researcher aims at creating two groups of respondents or research participants who are similar to each other. One group is then exposed to a programme, intervention or treatment and the comparison or control group not. In all other aspects, apart from the intervention, the two groups are treated equally.

If the two groups were the same before intervention, then differences in measurements after exposure are more likely to be due to the treatment. For this design to be successful it is very important that participants be randomly assigned to the two groups.

#### 4.3 Methodology

Participants were randomly assigned to two groups: an experimental and a control group. The control group was given a conservative training programme that needed to be followed twice a week over a period of 12 weeks. The experimental group had to follow a more progressive and aggressive training programme. They followed three different training programmes (each with a 4-week duration) that progressively became more aggressive. The experimental group also completed the three different training programmes over a period of 12 weeks.

Pre-test and post-test measures were taken of each group and the experimental group had to complete questionnaires as well. To determine if the two groups were the same prior to intervention and if the interventions had the desired effect, the relevant test and control groups were compared in the following ways:

- The **Mann-Whitney** test was used to compare the experimental and control groups on all pre-test measurements in order to determine whether the two groups were similar before intervention.
- After intervention, the measurements were repeated (post-test). The two groups were once again compared by means of the **Mann-Whitney** test.
- To determine whether changes had taken place from the pre-test measurements to the post-test measurements within the groups, the scores within each group (the experimental group and the control group) were compared by means of the **Wilcoxon signed-rank test**.

#### 4.4. Statistical Analysis

The collected measurements were captured on a computer and analysed by means of the Statistical Package for the Social Sciences (SPSS).

##### 4.4.1 Descriptive Statistics

Descriptive statistics has been reported as a method for describing data in manageable and understandable forms (Babbie, 1992). Descriptive statistics presented within this study included the number of participants, minimum and maximum scores, mean scores and standard deviations. These descriptive statistics gives the reader an indication of the nature of the data on all variables measured for reference purposes.

- **Mean score:** The mean score is used to describe central tendency. The mean score is computed by adding up all the applicable values and dividing them by the number of cases. (Research methods, knowledge base; <http://www.socialresearchmethods.net/kb/statdesc.php>).
- **Standard deviation:** The Standard Deviation shows the relation that a set of scores has to the mean of the sample. (Research methods, knowledge base;

<http://www.socialresearchmethods.net/kb/statdesc.php>). It gives an indication of the distribution of data around the mean on all variables measured. The higher the standard deviation, the more the data is dispersed.

#### **4.4.2 Inferential Statistics**

Test hypotheses about differences in populations on the basis of measurements were made on samples of patients (Tabachnick & Fidell, 1996).

##### **4.4.2.1 Mann-Whitney Test**

The Mann-Whitney test is a non-parametric test that is used to determine whether two samples are equivalent and drawn from the same single population. (Wikipedia, [http://en.wikipedia.org/wiki/Mann-Whitney\\_U](http://en.wikipedia.org/wiki/Mann-Whitney_U)).

In our case the active and placebo groups were compared during the pre-test and again during the post-test. Ideally the two groups should be the same during the pre-test and differ in favour of the active group during the post-test.

##### **4.4.2.2 Wilcoxon Signed-rank Test**

The Wilcoxon signed-rank test is a non-parametric test that can be used to test two related samples or repeated measurements on a single sample. The Wilcoxon test involves comparisons of differences between measurements. It is often used to test the difference between scores of data collected before and after an experimental manipulation. (Wikipedia, [http://en.wikipedia.org/wiki/Wilcoxon\\_signed-rank\\_test](http://en.wikipedia.org/wiki/Wilcoxon_signed-rank_test)).

#### **4.5. Results**

Results of the analysis will be presented in the following order:

- Descriptive statistics for the experimental and control groups for pre-test and post-test data on all measurements
- Difference between the experimental and control groups on pre-test measurements
- Difference between the experimental and control groups on post-test measurements

- Difference between the pre-test and post-test measurements within the experimental group
- Difference between the pre-test and post-test measurements within the control group.

#### 4.5.1. Descriptive Statistics for the Two Groups on all Measurements

These results are included simply as frame of reference for the reader to see how the two groups performed on all the measurements. The results are presented in tables 4.1 to 4.9.

**Table 4.1: Descriptive Statistics per Group on Pre-test Measurements**

Group		N	Minimum	Maximum	Mean	Std. Deviation
Experimental	Age	18	18	57	33.00	10.347
	Weight (kg)	18	50	131	85.56	26.988
	Height (cm)	18	155	195	175.17	12.894
	BMI (kg/m <sup>2</sup> )	18	19	36	27.17	5.973
	Hrs worked / day	18	5	15	10.19	2.573
	Time spent driving (minute)	18	.4	1800.0	169.189	494.9721
	Valid N (listwise)	18				
Control	Age	14	22	56	37.43	11.015
	Weight (kg)	14	59	106	79.11	14.369
	Height (cm)	14	152	190	170.57	10.761
	BMI (kg/m <sup>2</sup> )	14	20	36	27.14	3.939
	Hrs worked / d ay	14	3	16	9.07	2.786
	Time spent driving (minute)	14	.0	1800.0	129.764	480.7294
	Valid N (listwise)	14				

**A statistically significant difference was found at the 5% level of significance between the experimental and control groups for pre-test transport/driving time.**

The transport/driving time for the experimental group was significantly higher than that of the control group. No statistically significant differences were found between the pre-test of the experimental and control groups for any of the other measurements.

It can thus be concluded that, as far as the rest of the pre-test measurements are



concerned, the two groups were very similar. The process of randomisation has thus ensured that the groups were as homogenous as possible at pre-test, ensuring an even spread of respondents across both groups. However, the difference in driving time could be explained by the fact that one respondent in the experimental group worked very far from home, while some of the respondents in the control group worked from home; thus increasing the difference between the two groups.

Time spent driving and the amount of hours worked per day were also recorded. This was done because it is believed that increased time spent sitting might contribute to chronic low back pain (McGill, 2002). When commuting to work, travel time becomes a factor, especially when travel time becomes prolonged, involving sitting for extended periods of time. Vibration also plays a role, especially when driving in a car and the whole body is exposed to vibration. This might also contribute to chronic low back pain.

These two factors have been identified as causative factors (Andersson, 1997; Jansen *et al.*, 2002; Laursen & Scibye, 2002; Leboeuf-Yde, 2004). When combined, they may theoretically cause more problems than when a patient is exposed to only one of these. However, the results from the study are not indicative in this regard because some respondents travel long distances to work while others work from home. Future research is required to provide answers on this topic.

Time spent working may also be regarded as a causative factor, especially when static postures are maintained for long periods of time as is the case with office workers and computer personnel. The mean score for the amount of hours worked in the experimental group was 10.19 hours per week and 9.07 hours per week for the control group. This is more than the average work day of 8 hours per day. However, the high level of importance placed on work and returning to work in cases of low back pain have been discussed in detail in chapter 2.

Keeping patients away from work is not a sensible option. Work station modification and patient education have to play an important role in minimising strain placed on the patients with chronic low back pain to prevent absenteeism due to chronic low back pain.

**Table 4.2: Descriptive Statistics per Group on Pre-test and Post-test Measurements**

Group		N	Minimum	Maximum	Mean	Std. Deviation
Experimental	VAS Pain Score Pre-test	18	12	86	54.44	18.231
	VAS Pain Score week 4	12	5	72	33.92	23.899
	VAS Pain Score week 8	11	5	43	19.27	11.585
	VAS Pain Score week 12	11	1	63	17.00	18.746
	Tampa Scale Pre-test	18	5	15	8.28	2.296
	Tampa Scale week 4	12	4	12	8.25	2.006
	Tampa Scale week 8	11	4	10	7.27	1.849
	Tampa Scale week 12	11	4	10	6.82	1.940
	Valid N (listwise)	11				
	Control	VAS Pain Score Pre-test	14	25	84	52.57
VAS Pain Score week 4		13	10	64	30.77	14.313
VAS Pain Score week 8		10	5	63	28.80	21.364
VAS Pain Score week 12		10	2	35	13.40	11.462
Tampa Scale Pre-test		14	6	11	8.36	1.151
Tampa Scale week 4		13	4	12	8.08	2.019
Tampa Scale week 8		10	5	12	8.40	2.119
Tampa Scale week 12		10	4	14	7.30	2.830
Valid N (listwise)		10				

**Statistically significant differences were found at the 5% level of significance between the following pre-test and post-test variables within the experimental group.**

- VAS pain score: The pre-test measurement was significantly higher than the post-test measurement.

This shows a favourable result for the present study, as a lower score shows an improvement in pain levels. It has been shown that pain elimination is not a primary goal in the treatment of chronic low back pain (Staal *et al.*, 2003; Staal *et al.*, 2005). But the fact that the VAS pain score in the present study has decreased significantly within the experimental group demonstrates that an aggressive-progressive exercise programme is effective in treating pain associated with chronic low back pain, especially since the VAS score is below 30.

Pain was not completely eliminated in the experimental group (VAS pain score = 17.00) but this coincides with other studies that report that pain elimination might not be realistic in chronic low back pain due to the recurrence rate of low back pain (Shirado *et al.*, 2005; Staal *et al.*, 2005).

**Statistically significant differences were found at the 5% level of significance between the following pre-test and post-test variables within the control group.**

- VAS pain score: The pre-test measurement was significantly higher than the post-test measurement.

The control group also showed good improvement in their pain score. It has been proven that conservative exercise programmes are effective in treating low back pain (Richardson *et al.*, 1999; Hides *et al.*, 2001). Thus, the results in the present study confirm this view. However, conservative programmes will not necessarily cause the improvement in the overall functional status that the more aggressive programmes might have.

There was no significant difference between the post-test scores of the control and experimental groups. However, even if functional status improvement is regarded as being more important than pain levels, pain should still be addressed as a primary outcome goal. It has been reported by Meyer (2007) that poorly controlled pain remains a world-wide problem. In 1998 a survey indicated that about 22% of patients in primary care reported persistent pain over the past year. They believed that their pain was not treated properly (Gurejo *et al.*, 1998; Meyer, 2007).

The importance of pain has increased tremendously over the past decade. Chronic pain as a whole, which includes chronic low back pain, is now seen as a disease itself and not only as a symptom. It has also been recognised as the so-called 'fifth vital sign' and should be monitored with the same vigilance as blood pressure, temperature, pulse and respiratory rate (Meyer, 2007). Thus, pain management should be of primary concern and is just as important as functional status.

There was no significant difference of the post-test measurement between the control and experimental groups for the TAMPA scale, as well as from pre-test to post-test between either group. The mean score of either of the two groups was very high at pre-test or differed significantly, indicating that this particular cohort of chronic low back pain patients did not harbour a very high fear of movement to begin with. Both groups improved but not significantly. The experimental group improved more

than the control group, although not significantly. This result can partially confirm the success of the experimental group exercise programme.

However, it has been reported that very intensive multidisciplinary rehabilitation, such as more than a hundred hours is needed to achieve favourable results on those who are actively working (Kääpä *et al.*, 2006). It has been mentioned previously that all of the patients in the study were full working capacity adults and more active back school might have been more beneficial towards this variable. The experimental group received only 2.5 hours' worth of back school counselling.

**Table 4.3 : Descriptive Statistics per Group on Pre-test and Post-test Measurements (continued)**

Group		N	Minimum	Maximum	Mean	Std. Deviation	
Experimental	Oswestry Disability Index Pre-test	18	11	44	23.72	8.574	
	Oswestry Disability Index week 4	12	2	30	15.42	7.416	
	Oswestry Disability Index week 8	11	0	18	10.18	6.431	
	Oswestry Disability Index week 12	11	0	20	8.00	7.376	
	Functional Rating Index Pre-test	18	15	63	34.61	13.232	
	Functional Rating Index week 4	12	0	38	19.58	9.307	
	Functional Rating Index week 8	11	0	25	14.82	7.960	
	Functional Rating Index week 12	11	0	25	10.64	8.686	
	Valid N (listwise)	11					
	Control	Oswestry Disability Index Pre-test	14	10	34	20.07	7.731
		Oswestry Disability Index week 4	13	6	42	19.69	10.379
Oswestry Disability Index week 8		10	4	30	14.20	7.068	
Oswestry Disability Index week 12		10	2	20	11.00	6.200	
Functional Rating Index Pre-test		14	23	43	32.29	7.559	
Functional Rating Index week 4		13	13	48	27.00	8.972	
Functional Rating Index week 8		10	10	33	20.50	6.060	
Functional Rating Index week 12		10	3	23	13.80	6.233	
Valid N (listwise)		10					

**Statistically significant differences were found at the 5% level of significance between the following pre-test and post-test variables within the experimental group:**

- Oswestry Disability Index and Functional Rating Index: The pre-test measurements were significantly higher than the post-test measurements.

This shows a favourable result for the present study, as a lower score shows an improvement in self-reported disability levels. A significant decrease in disability levels, as shown by the Oswestry Disability Index and the Functional Rating Index, demonstrates that an aggressive-progressive exercise programme may also be effective in decreasing levels of self-reported disability. Research has argued the importance of disability levels in chronic pain (Dionne *et al.*, 1995; Cherkin *et al.*, 1996; Waddell, 1996; Dionne *et al.*, 1997; Pflingsten *et al.*, 1997; Epping *et al.*, 1998; Thomas *et al.*, 1999; Linton *et al.*, 2000; Pincus *et al.*, 2002; Staal *et al.*, 2003; Staal *et al.*, 2005), as well as the need to reduce it (Ashburn & Staats, 1999; Sanders *et al.*, 1999; Simmonds & Dreisinger, 2003; Sanders *et al.*, 2005).

Linton *et al.* (2005) reported that it is possible to even prevent the development of pain-related disability by providing specific interventions, which focus on the psychosocial and functional troubles that patients find problematic. Any type of exercise programme that can reduce disability levels may be considered a worthwhile treatment modality, especially an aggressive-progressive type of exercise programme that may improve functional status in the long-term (Manniche *et al.*, 1991; Johannsen *et al.*, 1995; Petersen *et al.*, 2002; Ostelo *et al.*, 2003). This, in conjunction with high-intensity back school, has an overall improvement in low back outcomes.

**Statistically significant differences were found at the 5% level of significance between the following pre-test and post-test variables within the control group:**

- Oswestry Disability Index and Functional Rating Index: The pre-test measurement was significantly higher than the post-test measurement.

The control group also showed a good outcome in terms of disability variables. This shows that a conservative exercise programme is very effective in improving disability due to low back pain. It has been reported that physical exercise is recommended to prevent absence due to back pain and the occurrence or duration of further back pain episodes (Burton, 2005). Exercise therapy for chronic low back pain is recommended by several guidelines (Spitzer *et al.*, 1987; Albright, 2001; Hayden *et al.*, 2005; Krismer & Van Tulder, 2007).

Impaired function of trunk muscles has been shown to have a close correlation with the pathogenesis of chronic low back pain (Shirado *et al.*, 1992; Shirado *et al.*, 1995a; Ito *et al.*, 1996). The main purpose of therapeutic exercise is to strengthen trunk muscles and improve trunk flexibility (Shirado *et al.*, 1995b). Stabilisation exercises differ from general exercises by being more body-specific, and requiring more attention and precision from the patient (Bergmark, 1989a). This proves that a conservative exercise programme is still effective in improving levels of disability.

In both the Oswestry Disability Index as well as the Functional Rating Index the mean scores in the experimental group were lower than in the control group, but not significantly better. This could argue a better improvement in the experimental group. It could be due to the nature of the back school, which was more intensive than in the control group. The one-on-one sessions used in the experimental group could have had more of an improvement. The control group received only the textbook to read through on their own. The experimental group received the textbook as well as one-on-one attention about the contents. This type of counselling has been reported to be effective in the treatment of chronic low back pain (Tulder *et al.*, 2001).

However, the amount of back school intervention used in the present study was not in line with previously reported research. The Swedish approach of four sessions per week lasted around 30 minutes each (Heymans *et al.*, 2006). High intensity back schools used a twice-a-week approach for eight weeks. These consisted of 16 sessions, each lasting an hour (Vlaeyen *et al.*, 1995). It has also been reported that only very high-intensity multidisciplinary rehabilitation (>100 hours) can be effective for chronic low back pain (Guzman *et al.*, 2001).

The present study used 10 sessions lasting approximately 10 minutes each. It was expected that shorter sessions would not lead to boredom and lack of interest on the part of the patients. It has been reported previously that educational and exercise sessions require keen concentration and active participation, especially in those patients who work full-time (Kääpä *et al.*, 2006).

Patients in the experimental group expressed their gratitude on the educational sessions not being lengthy in duration, especially at the end of a long working day. Future research has to find a balance between sufficient time to convey enough information to not cause mental fatigue in patients, especially in those of full working capacity.

**Table 4.4: Descriptive Statistics per Group on Pre-test and Post-test Measurements (continued)**

Group		N	Minimum	Maximum	Mean	Std. Dev
Experimental	FABQ Pre-test Scale 1	18	0	26	9.61	8.052
	FABQ Pre-test Scale 2	18	0	20	12.11	5.940
	FABQ Pre-test Total	18	2	42	21.72	10.260
	FABQ Scale 1 week 4	12	0	19	6.58	5.616
	FABQ Scale 2 week 4	12	2	20	12.42	4.757
	FABQ Total week 4	12	0	36	19.00	8.975
	FABQ Scale 1 week 8	11	0	22	5.73	7.001
	FABQ Scale 2 week 8	11	0	15	8.09	5.375
	FABQ Total week 8	11	3	29	14.09	9.049
	FABQ Scale 1 week 12	11	0	24	6.27	8.113
	FABQ Scale 2 week 12	11	0	12	4.09	3.727
	FABQ Total week 12	11	1	24	10.36	7.877
	Valid N (listwise)	11				
Control	FABQ Pre-test Scale 1	14	0	28	12.64	10.035
	FABQ Pre-test Scale 2	14	1	24	14.29	5.663
	FABQ Pre-test Total	14	6	45	26.93	10.923
	FABQ Scale 1 week 4	13	0	30	9.69	9.277
	FABQ Scale 2 week 4	13	2	21	12.77	5.510
	FABQ Total week 4	13	11	51	22.46	11.801
	FABQ Scale 1 week 8	10	0	23	10.70	8.111
	FABQ Scale 2 week 8	10	3	18	11.10	4.358
	FABQ Total week 8	10	9	38	21.40	9.336
	FABQ Scale 1 week 12	10	0	21	6.40	7.877
	FABQ Scale 2 week 12	10	0	19	7.00	6.716
	FABQ Total week 12	10	0	40	13.40	12.176
	Valid N (listwise)	10				

**Statistically significant differences were found at the 5% level of significance between the following pre-test and post-test variables within the experimental group:**

- FABQ Scale 2: The pre-test measurement was significantly higher than



the post-test measurement.

In the Fear Avoidance Beliefs Questionnaire the second scale measured fears regarding work and its influence on pain and disability (Swinkels-Meewisse *et al.*, 2003). Working status is an important factor in the management of chronic low back pain (Pincus *et al.*, 2002). Work absenteeism plays an important role in the recovery of patients, and recurrences in pain can lead to recurrences in work absenteeism, as well as restrictions in usual activities (Hildebrandt *et al.*, 2004; Katz, 2006). Especially when pain becomes long-standing it can lead to severe disability due to work absence and severe social consequences such as isolation (Punnett *et al.*, 2005). It is because of this that return to work has been recommended as an important goal in the management of chronic low back pain (Staal *et al.*, 2005).

However, in the present study, all of the patients were of full working capacity and nobody was absent from work for any amount of time due to their back pain. Staal *et al.* (2005) have proposed that while working, the disabled worker realises that he or she is still active despite being in discomfort. Being at work, in a partial or full capacity, draws the attention of the disabled worker away from negative issues such as pain and helps to decrease the focus of the disablement. This could provide an explanation to why the patients in the present study did not present with high levels of disability to start with, and why they are all of full working capacity status, even though many of them have been suffering from low back pain for many years.

Even after treatment the patients in the aggressive-progressive exercise group reported that they felt less fear about activities in their working lives and that they were able to perform certain activities with more confidence and less anticipation for developing pain. It has been reported that pain relief is not necessary to resume work in any case (Lindstrom *et al.*, 1995; Crombez *et al.*, 1999; Van Tulder *et al.*, 2000). This suggests a more subjective outlook on pain experienced by the patients. It could be argued that the programme instilled more confidence in the patients regarding their day-to-day working activities.

- FABQ Total: The pre-test measurement was significantly higher than the post-test measurement.

Fear of pain, as measured by the Fear Avoidance Beliefs Questionnaire, during an episode of back pain is related to chronic pain status at follow-up (Gatchel *et al.*, 1995; Klenerman *et al.*, 1995; MacFarlane *et al.*, 1999), making fear avoidance an important factor in the treatment of low back pain simply due to the fact that back pain can be triggered by psychological factors such as a fear of pain or illness (Carragee *et al.*, 2000).

It is reported that patients who perceive pain in a threatening, catastrophic manner are much more likely to experience pain-related fear and anxiety, and consequently to engage in escape or avoidance behaviours to situations that they perceive to be potentially harmful (Thomas & France, 2007). Over time avoidance of these activities of daily living that are perceived to increase pain or risk for re-injury, is repeatedly reinforced by anxiety reduction. Hence, pain-related fear and anxiety ultimately contribute to symptoms of disuse and disability (Thomas & France, 2007).

It has also been suggested that fear avoidance beliefs for physical activity follow the same clinical pattern as pain and related disability (Grotle *et al.*, 2006). This even has a physical component in that guarded movements and hyperactivity in the lumbar paraspinal muscles are correlated with pain-related fear (Maffey-Ward *et al.*, 1996; Main & Watson, 1999). It can thus be argued that pain and fear avoidance behaviour will contribute to disability along with work absenteeism, causing a cycle that will ultimately lead to severe forms of disability and activity restriction.

Exercise has an important role to play in the management of fear avoidance behaviour. It has been suggested that exercise could have a therapeutic effect by improving physical function impaired by chronic back low pain, improving back pain intensity and improving disability through a process of desensitisation of fears and concerns, altering pain attitudes and beliefs (Rainville *et al.*, 2000).

This was the goal of the back school used in the present study in conjunction with the exercise programme, namely to eliminate fears regarding pain, as well as any resulting disability caused by pain and its related fear. It has been shown that fear avoidance beliefs are significantly more reduced in patients who are provided with

cognitive intervention and exercise (Brox *et al.*, 2003). The result from the current study confirms this finding. An aggressive-progressive exercise programme, in conjunction with high-intensity back school, can help decrease pain-induced fear avoidance behaviour in those suffering from chronic low back pain. It will ultimately decrease overall disability caused by chronic low back pain.

**Statistically significant differences were found at the 5% level of significance between the following pre-test and post-test variables within the control group:**

- FABQ Scale 1, FABQ Scale 2 and FABQ Scale Total: The pre-test measurements were significantly higher than the post-test measurements.

The control group programme was also very effective in decreasing the amount of fear avoidance the patients experienced. As has been reported previously, exercise has an important role to play in the management of fear avoidance behaviours. It has been suggested that exercise could have a therapeutic effect by improving physical function impaired by chronic back low pain, improving back pain intensity and improving disability through a process of desensitisation of fears and concerns, altering pain attitudes and beliefs (Rainville *et al.*, 2000).

This was the goal of the back school used in the present study in conjunction with the exercise programme, namely to eliminate fears regarding pain, as well as any resulting disability caused by pain and its related fear. It has been shown that fear avoidance beliefs are significantly more reduced in patients who are provided with cognitive intervention and exercise (Brox *et al.*, 2003). However, the back school used in the control group was very low intensity. Patients were only given the textbook to read on their own. Some of the patients admitted that they never read through the book. This could question the effectiveness of the back school, as the control group also showed significant improvements in not only fear avoidance beliefs, but also in disability variables.

The reason for the improvement in the variables of the control group could be that the exercise programme has more than just a physical strengthening effect. It could be that the patients can observe their own physical improvement over the 12 weeks

and then realise that they are more capable than they previously believed. This could then lead to more confidence in previously avoided activities in both personal and occupational settings. The back school could thus reaffirm their confidence in line with their own observations of feeling less scared to move more and be more active.

**There was no significant difference found between the experimental and control groups at post-test level for the Fear Avoidance Beliefs Questionnaire (FABQ) for any of the scales.**

This result questions the effectiveness of the high-intensity back school used in the experimental group. It was expected to be more significantly effective than the low-intensity back school used in the control group. This again only partially confirms the hypothesis of the study because the experimental group was only slightly more effective. The high-intensity back school was certainly effective, as was shown by the results from pre-test to post-test. In the high-intensity back school approach the confrontational method to pain management was used, as suggested by Lethem *et al.*, (1983).

However, an exercise intervention method uses this approach by default, as it challenges the fear of movement that patients could have (Vlaeyen & Linton, 2000). The back school could reaffirm this new belief and confidence to move and to be active. This could be the reason why there was a small difference between the experimental and control groups' back schools. The exercise programmes achieved most of the effect, as it challenged the patients to be active in a way that they did not think possible. To achieve a better result from the back school it might be more effective to identify patients who would benefit more from a cognitive intervention strategy, as proposed by Kääpä *et al.* (2006).

**Table 4.5: Descriptive Statistics for the Experimental Group on Questionnaire Measurements**

Group		N	Minimum	Maximum	Mean	Std. Deviation
Experimental	Cycling Program 1	11	6	12	8.09	2.212
	Cycling Program 2	11	6	14	9.00	2.864
	Cycling Program 3	11	6	17	9.91	3.754
	Lat Pulldown Program 1	11	6	12	9.18	2.183
	Lat Pulldown Program 2	11	6	14	10.27	2.936
	Lat Pulldown Program 3	11	7	17	11.36	2.908
	Cable Exercise Program 1	11	5	13	9.18	2.316
	Cable Exercise Program 2	11	6	18	11.64	4.007
	Cable Exercise Program 3	11	6	13	9.36	2.580
	Valid N (listwise)	11				

Tables 4.5 and 4.6 measured the results from the Borg Rate of Perceived Exertion(RPE) scale. None of the results differs significantly. At best there is a small increase from the first exercise through to the third exercise. This suggests that there were small increases in intensity between each successive exercise, except for the Cable Exercise 2, which achieved a higher score than Cable Exercise 3. This indicates that it would have been better to have had Cable Exercise 2 in the third programme rather than the second programme. These results show a progressive increase in exercise intensity between the programmes. The exercises were thus ideal for the programmes in their progression stages.

**Table 4.6: Descriptive Statistics for the Experimental Group on Questionnaire (continued)**

Group		N	Minimum	Maximum	Mean	Std. Dev.
Experimental	Side Bridging Program 1	11	7	20	12.09	3.859
	Side Bridging Program 2	11	7	20	13.27	3.875
	Side Bridging Program 3	11	9	20	14.64	3.384
	Hip Lifts Program 1	11	9	14	11.73	1.489
	Hip Lifts Program 2	11	10	19	13.00	2.966
	Hip Lifts Program 3	11	9	15	12.45	1.809
	Alt Superman Program 1	11	7	15	10.82	2.676
	Alt Superman Program 2	11	7	14	11.09	2.427
	Alt Superman Program 2	11	7	15	11.64	2.693
	Abdominal Crunch Program 1	11	9	16	12.18	2.089
	Abdominal Crunch Program 2	11	9	16	12.91	1.921
	Abdominal Crunch Program 3	11	12	18	14.82	1.888
	Valid N (listwise)	11				

The results from Table 4.6 show a very similar tendency as Table 4.5, because there is a small increase in exercise intensity throughout the stages of progression. Only Hip Lifts Programme 2 showed a higher score than the exercise of the third programme (Hip Lifts Programme 3). Thus, it would have been better to have had that exercise rather in the third programme, and Hip Lifts Programme 3 in the second programme.

The concept of using an RPE scale has been investigated in the past. Barker *et al.* (2003) used an RPE scale to measure the intensity of aerobic exercise in patients with chronic low back pain when performing exercise in a hydrotherapy pool. They found that at workloads sufficient to induce an aerobic training response and still safe for the patients, the rate of perceived exertion was an accurate predictor of exercise intensity. Unfortunately it is difficult to compare the results from the present study with those from the Barker *et al.* (2003) study, as they used heart rate to compare with perceived intensity.

They also compared it with aerobic fitness levels. The present study did not measure heart rate at any stage during the study and it was never sought to increase the heart rate of the patients in order to gain aerobic fitness. The rate of perceived exertion scale was only used to measure the perceived intensity of the performed exercises in order to gain insight into their difficulty in performing for those with chronic low back pain.

Wallbom *et al.* (2002) also used physiological parameters to compare with rate of perceived exertion scales. It was found that a percentage of maximum heart rate is significantly related to self-reported pain and disability, as well as age. It is suggested that perceived exertion in populations with disabling back pain is not highly correlated with physiologic effort, as other factors such as pain may influence effort rating (Wallbom *et al.*, 2002). Again physiological parameters were not measured in the present study to be able to compare it with the other findings.

However, just like in the Wallbom *et al.* (2002) study, it was found in the present study that physiological exertion was not necessarily the limiting factor, but pain probably was more so. It is thus unclear if patients with chronic low back pain rate the intensity of the exercises according to physiological effort or pain. Much more research will be needed in future if a rate of perceived exertion scale is to be used for patients with chronic low back pain.

#### **4.5.2 Frequency, Intensity and Duration**

In terms of frequency, intensity and duration it is recommended that for frequency 2-3 times per week is sufficient (Manniche *et al.*, 1991; Oldridge & Stoll, 1997; Perkins & Zipple, 2003). Both the control and experimental groups performed their programmes twice per week in line with the recommendation. However, it is suggested that in early rehabilitation exercises should be performed daily with decreasing frequency as exercise tolerance increases. None of the patients in the study had ever participated in a low back rehabilitation programme up to the point of the intervention. A case could be made for rather performing the exercises daily, but it was decided to keep to two sessions per week to ensure supervision. Patients were not able to attend every day and the decision to supervise was decided to be more important.

The intensity of the exercise programmes, specifically for those with chronic low back pain, is more difficult to determine, as clear instructions are difficult to obtain. Available instructions usually suggest that the exercise prescribed should be more intense than that normally prescribed for back patients (Perkins & Zipple, 2003). The



reason for this could be the necessity to increase functional ability, as those with chronic low back pain tend to become more disabled with time (Bergquist-Ullman & Larsson, 1977). One set of instructions, described as the Delorme method for intensity selection, suggests selecting resistance that allows 20-30 repetitions with proper neuromuscular control in a pain-free or minimal painful range of motion (Perkins & Zipple, 2003). Initially this will increase endurance and control of movement. As the person progresses, resistance should be increased while the number of repetitions decreased to 8-12, which is comparable with ACSM strength training guidelines (ACSM, 2000).

Self-selected intensity or exercise to pain tolerance often leads to inadequate exercise levels. Although pain might not improve for several months in many patients, an intensive exercise programme may result in greater functional and psychological benefits than a less aggressive approach (Perkins & Zipple, 2003).

Others also suggest a quota approach, in which exercise intensity is prescribed to prevent under-exercising (Lindstrom *et al.*, 1992; Linton, 1994; Rainville *et al.*, 1997). This lends support for the use of a more aggressive exercise programme as used in the present study to focus on more issues relating to chronic low back pain than only subjective pain levels.

Also, the present study attempted to use the Borg RPE scale to measure the intensity of the experimental group. The results show slight progressing in most exercises throughout the three training programmes. It shows sufficient exercise intensities to promote a training effect, but was not too strenuous at any stage that the danger of injury existed.

However, the present study failed to measure the control group by means of the RPE scale, which will place a limitation on the results of the experimental group because the two groups cannot be compared on this variable. Future studies should further investigate the use of this scale in patients with chronic low back pain and to determine whether this is a viable scale to use in rehabilitation for this population group.

In terms of duration it is recommended that a training session lasts for between 20-30 minutes of aerobic exercise and weight training of 30-60 minutes (ACSM, 2000). However, it was felt that this recommendation is too lengthy for rehabilitation. The entire training session of the control group lasted only 35 minutes. The group's only form of aerobic training was in the warm-up exercise that lasted for only five minutes.

The experimental group also did only five minutes, but their cycling increased in intensity throughout the entire programme. The rationale behind this was that the goal of the rehabilitation programme was to first and foremost help the patients with their back pain. It was instructed to them to initiate exercise programmes after the intervention period consisting of longer duration and higher intensity. As the patients started to feel improvement during the study, many of them became more motivated to start with more difficult exercise programmes. All patients received guidelines of exercises to perform after the intervention period.

**Table 4.7: Descriptive Statistics for the Experimental and Control groups (continued)**

Group		N	Minimum	Maximum	Mean	Std. Dev.	
Experimental	Pre-test Straight leg rise Neural Tension Left	20	1	2	1.90	.308	
	Pre-test Straight leg rise Neural Tension Right	18	1	2	1.83	.383	
	Pre-test Straight leg rise Hamstring Degree Left	18	46	110	67.17	16.660	
	Pre-test Straight leg rise Hamstring Degree Right	18	22	90	65.28	16.215	
	Post-test Straight leg rise Neural Tension Left	11	1	2	1.64	.505	
	Post-test Straight leg rise Neural Tension Right	11	1	2	1.82	.405	
	Post-test Straight leg rise Hamstring Degree Left	11	65	92	79.55	8.335	
	Post-test Straight leg rise Hamstring Degree Right	11	70	101	82.82	10.685	
	Valid N (listwise)	11					
	Control	Pre-test Straight leg rise Neural Tension Left	15	1	2	1.73	.458
		Pre-test Straight leg rise Neural Tension Right	14	1	2	1.57	.514
		Pre-test Straight leg rise Hamstring Degree Left	14	44	98	74.29	15.107
		Pre-test Straight leg rise Hamstring Degree Right	14	39	87	68.36	15.540
		Post-test Straight leg rise Neural Tension Left	10	1	2	1.80	.422
		Post-test Straight leg rise Neural Tension Right	10	1	2	1.70	.483
Post-test Straight leg rise Hamstring Degree Left		10	68	94	83.50	7.778	
Post-test Straight leg rise Hamstring Degree Right		10	69	92	79.80	8.728	
Valid N (listwise)		10					

**Statistically significant differences were found at the 5% level of significance between the following pre-test and post-test variables within the experimental group:**

- Straight Leg Rise Hamstring Degree Left and Right: The post-test measurements were significantly higher than the pre-test measurements.

It has been shown that patients with a history of low back pain have reduced hamstring flexibility (Biering-Sorensen, 1984). The aggressive-progressive exercise programme contained stretching exercises that were selected to improve hamstring flexibility. This was selected because of the effect of the hamstring muscles on low

back posture. Tight hamstrings are prominent in postures such as flat-back and sway-back postures (Kendall *et al.*, 1993). These types of postures have been associated with back pain in the past, and by stretching these muscles pressure on the low back may be alleviated by correcting possible imbalances due to postural faults (Kendall *et al.*, 1993). However, the limited available data does not support the view that greater flexibility of the spine prevents injury, but it is important to maintain adequate flexibility at the hips and knees for lifting (Perkins & Zippel, 2003). This supports the motivation for performing stretches that involve not only the hamstrings, but also the quadriceps muscle group, as well as the buttocks and hips, as was performed by the experimental group.

**Statistically significant differences were found at the 5% level of significance between the following pre-test and post-test variables within the control group:**

- Straight Leg Rise Hamstring Degree Right: The post-test measurement was significantly higher than the pre-test measurement.

There was a decrease in both legs in this group for this value. The control group performed a hamstring stretching exercise that was much simpler than that of the experimental group. The exercise also stayed the same throughout the 12 week intervention period. The exercise was selected due to its conservative non-threatening application. However, this result indicates the shortcoming of this exercise in terms of being insufficient when strengthening exercises are also involved.

The control group performed many exercises than involve and ultimately strengthen the hamstring muscle group (e.g. hip lifts), just like the experimental group. Skeletal muscles adapt their length and stiffness according to the functional demands to which they are regularly submitted. This modification of muscle stiffness and length induced by resistance training may alter the joint stiffness and theoretically change the joint resting position (Ocarino *et al.*, 2008). This indicates the necessity of a more aggressive type of stretching exercise to ensure that the hamstring muscle group retains its mobility. As has been discussed, it is very important to maintain the

flexibility of the hamstrings to ensure functional activities are performed with maximum ease (Perkins & Zipple, 2003).

**Statistically significant differences at the 5% level of significance were found between the post-test measurements of the experimental and control groups of the following:**

- Straight Leg Rise Hamstring Degree Right: The post-test value of the experimental group was significantly higher than that of the control group.

This result is to be expected due to the fact that the experimental group had many more exercises that were selected to increase the flexibility of the hamstring muscles. The reason that this result was only found on the right side could be due to the dominant side being preferred by the patients and thus more effort was applied. At the post-test all of the patients remaining in the study reported being right side dominant (89%).

**Table 4.8: Descriptive Statistics for the Experimental and Control groups (continued)**

Group		N	Minimum	Maximum	Mean	Std. Dev.
Experimental	Pre-test Slump	18	1	2	1.39	.502
	test Left					
	Pre-test Slump	18	1	2	1.17	.383
	test right					
	Post-test Slump	11	1	2	1.36	.505
Control	test Left					
	Post-test Slump	11	1	2	1.36	.505
	test Right					
	Valid N (listwise)	11				
	Pre-test Slump	14	1	2	1.43	.514
	test Left					
	Pre-test Slump	14	1	2	1.29	.469
test right						
Post-test Slump	10	1	2	1.20	.422	
test Left						
Post-test Slump	10	1	2	1.30	.483	
test Right						
Valid N (listwise)	10					

The slump test was performed at pre-test and at post-test evaluation. This test is designed to place the Sciatic nerve under increasing levels of tension (Majlesi *et al.*, 2008). The test applies traction to the nerve roots by incorporating spinal and hip

flexion into leg raising and warns of the presence of nerve root compression (Majlesi *et al.*, 2008). However, the results from the slump test are too varied and no consistent pattern emerged; thus, no conclusion can be drawn other than that there was no indication of nerve root compression.

The value of the slump test was that it was used to assist in eliminating any possible 'red flag' conditions. No patient tested either at pre-test or post-test presented with any symptoms such as sciatic pains or worsening of back pain. All of the discomfort felt, only indicated hamstring tightness or neural tension. All tension was relieved when cervical extension was added. Relief or partial relief following cervical extension indicates a normal response to the slump test. It may thus be considered to be a neurogenic response (Walsh *et al.*, 2007). No patient was unable to have the test performed on him and none showed lasting symptoms afterwards.

**Table 4.9: Descriptive Statistics for the Experimental and Control Groups (continued)**

Group		N	Minimum	Maximum	Mean	Std. Deviation	
Experimental	Pre-test Modified Sorenson Test	18	8	180	69.17	46.732	
	Post-test Modified Sorenson Test	11	31	240	138.82	73.929	
	Pre-test Side Bridging Left	18	0	92	30.61	23.553	
	Pre-test Side Bridging Right	18	2	96	34.78	25.917	
	Post-test Side Bridging Left	11	30	180	73.00	44.215	
	Post-test Side Bridging Right	11	17	180	71.45	44.098	
	Pre-test 60 degree Flexor	17	19	180	89.71	66.987	
	Post-test 60 degree Flexor	11	39	240	151.09	70.438	
	Valid N (listwise)	11					
	Control	Pre-test Modified Sorenson Test	14	0	114	47.57	30.686
		Post-test Modified Sorenson Test	10	25	180	112.00	57.417
		Pre-test Side Bridging Left	14	5	49	27.71	11.964
		Pre-test Side Bridging Right	14	6	48	27.14	12.799
		Post-test Side Bridging Left	5	13	43	29.20	13.349
Post-test Side Bridging Right		5	13	48	28.60	12.896	
Pre-test 60 degree Flexor		14	21	114	71.64	31.402	
Post-test 60 degree Flexor		10	45	240	114.60	63.479	
Valid N (listwise)		5					

**Statistically significant differences were found at the 5% level of significance between the following pre-test and post-test variables within the experimental group:**

- Modified Sorenson Test: The pre-test measurement was significantly lower than the post-test measurement in the experimental group.

This was an excellent result for the present study. Research demonstrated the importance of the low back extension musculature in low back pain research and also predicted low back pain prevalence (Biering-Sorensen, 1984; Adams *et al.*, 1999; Sjolie & Ljunggren, 2001; McGill, 2002; Demoulin *et al.*, 2004). The fact that this group was able to increase their back extensor endurance values has important



implications for the future. For one, it has shown that the patients in the experimental group decreased their chances for future episodes of low back pain, as described by Biering-Sorensen (1984). By increasing their extensor muscle endurance, the patients gained protection against external loads by increasing the load-bearing ability of the extensor muscle.

A positive aspect of the aggressive-progressive group was that it increased the extensor muscle endurance and the overall functional ability of the patients, as well as decreasing pain levels. These three aspects have to be considered the three most important outcomes when dealing with chronic low back pain. All three aspects have been described in the literature in great detail, and have to be considered in both research and the private setting. However, all of the involved musculature have to be considered in low back pain research (extensor, flexor and lateral musculature) and not only the extensor group (McGill, 2002).

Also, at pre-test many patients reported that they had stopped because of pain, but those who stopped at the post-test reported fatigue as the limiting factor. As described by others, pain may be the reason why patients choose to terminate the test and not necessarily because of muscular fatigue (Biering-Sorensen, 1984; Mannion & Dolan, 1994; Latikka *et al.*, 1995; Moreland *et al.*, 1997; Latimer *et al.*, 1999). This finding was also reported in the present study. Pain was still reported by some patients at post-test, but pain reported was minimal and not stated as the reason for terminating the test. Only when the patients decreased their fear avoidance beliefs as well, were they able to sustain the test beyond pain levels and to the point of muscular fatigue.

It has been reported that shorter holding time in patients with a history of low back pain may reflect test fear avoidance behaviour (Waddell *et al.*, 1993). No patients in either of the two groups reported increased levels of pain or discomfort when they completed the extension test. It can also then be argued that pain apprehension and not true reflected levels of pain were the limiting factors in the present study. It has been suggested that those with a history of low back pain tend to have shorter holding times (Ropponen *et al.*, 2005). It has been suggested that those with daily low back pain are much more likely to stop the test due to pain. The clinical

implication would suggest that the isometric back extension endurance test might reflect current back symptoms and pain tolerance of those with daily low back pain history more than it serves as a measure of physical capacity such as isometric back endurance (Ropponen *et al.*, 2005).

Subjective pain experience again plays an important role. Thus, at post-test patients could extend themselves without the fear of causing increased damage to themselves. This phenomenon was also reported to the patients who then realised that they were not limited by their current pain levels but rather by fear avoidance beliefs. This also served as proof to the patients that they were capable of more functional capacity than they initially believed even though minimal levels of pain were still present.

The aggressive-progressive exercise programme was able to decrease pain levels significantly, and increase the strength endurance of the extensor muscles and the functional ability of the patients. This is in accordance with other researchers who have suggested that patients with low back pain should gain from somewhat higher intensity of exercise than commonly used (Mayer *et al.*, 1985).

**Statistically significant differences were found at the 5% level of significance between the following pre-test and post-test variables within the control group:**

- Modified Sorenson Test: The pre-test measurement was significantly lower than the post-test measurement in the control group.

The control group performed exercises that are traditionally done for low back pain, especially McKenzie-based extension movements. These types of back extension movements are often suggested for strengthening the erector spinae muscles (D'Orazio, 1999). Recommended exercises included the alternative superman exercise (Perkins & Zipple, 2003), which was performed by both the control and experimental group. This exercise has been suggested to produce low levels of spine compression and is relatively safe for those suffering from chronic low back pain (Perkins & Zipple, 2003). Because the control group performed this exercise in

its most basic form (on all fours), this version is probably safer than the exercise versions performed by the experimental group.

**No significant difference was found between the post-test values of the control and experimental groups.**

The mean score in the experimental group was higher than that of the control group. This again partially confirmed the hypothesis of the study in which the experimental variable was higher than that of the control, although not significantly. However, this result could have been expected, as both groups concentrated on performing extension based exercises that were expected to increase this variable. The improved difference seen in the experimental group may be attributed to the other exercises included in the programme. These had more of a combined effect than the extension-based exercises alone.

The control group performed exercises that are traditionally done for low back pain, especially McKenzie-based extension movements. These types of back extension movements are often suggested for strengthening the erector spinae muscles (D’Orazio, 1999). Recommended exercises included the alternative superman exercise (Perkins & Zipple, 2003), which was performed by both the control and experimental group. This exercise has been suggested to produce low levels of spine compression and is relatively safe for those suffering from chronic low back pain (Perkins & Zipple, 2003). Because the control group performed this exercise in its most basic form (on all fours), this version was probably safer than the exercise versions performed by the experimental group.

However, the results from the extension exercises have to be interpreted with caution. It has been reported that when the time to endurance limit is used as the measure of muscle fatigability, the termination of the test is strongly dependant on the patient’s motivation and current levels of low back pain rather than actual muscle fatigability (Ropponen *et al.*, 2005). This is where fear avoidance behaviour plays an important role, because it may determine the extent to which a patient is prepared to exert himself.

Also, chronic low back pain patients active in working life do not tend to show any impairment in paraspinal muscle function or fatigability during dynamic endurance tasks (Kankaanpää *et al.*, 2005). As all of the patients in the present study were of full working capacity, it can be assumed that the main motivator for test termination can be related to fear avoidance behaviour and apprehension, and not necessarily pain.

**Statistically significant differences were found at the 5% level of significance between the following pre-test and post-test variables in the experimental group:**

- Side Bridging Left and Right: The post-test measurement is significantly higher than the pre-test measurement in the experimental group.

This result shows the success of the progression of the side bridging exercises in the experimental group. The importance of the lateral musculature has been shown in the literature (McGill, 1991; McGill, 1992; Juker *et al.*, 1998), especially since the obliques seem to be involved in stabilisation as well as movement (Belen'kii *et al.*, 1967; Bouisset & Zattara, 1981; Zattara & Bouisset, 1988; Aruin & Latash, 1995; Todorov & Jordan, 2002; Granada & England, 2006). The side bridging exercise has been shown to optimally activate the internal and external obliques, while placing minimal loads on the lower back but still provides enough stimuli to effectively train the muscles (Juker *et al.*, 1998; McGill, 1998; McGill, 2002).

The side bridging exercise also has the added advantage of training the quadratus lumborum, which is an important lumbar stabiliser (Perkins & Zippel, 2003). For example, during the second progression version of the side bridging exercise in which the patients performed the exercise with straight legs and holding the position static, the lumbar compression was a modest 2500N. However, the quadratus lumborum and the oblique closest to the floor appeared to be active up to 50% of the maximal voluntary contraction (MVC) (McGill, 2002).

**There was no significant difference between the pre-test and post-test values for the left and right side bridging test in the control group.**

This result was expected, as the control group performed no exercises to strengthen the lateral musculature. Due to the nature of the importance of the lateral musculature exercises to strengthen this component had to be included. Exercise with low levels of compressive force had to be included to achieve a total strengthening of all the important abdominal muscles.

**Statistically significant differences at the 5% level of significance of the following were found between the post-test measurements of the experimental and control groups:**

- Side Bridging Left and Right: The post-test values of the experimental group were significantly higher than those of the control group.

This is an important result due to the nature of the design of the programmes. The experimental group programme contained exercises that are used to strengthen the lateral musculature of the trunk, while the control group programme contained no exercises to strengthen this component. The different versions of the side bridging exercise used in the experimental group have been reported to be very effective exercises to strengthen the lateral trunk musculature with minimal compressive force penalty on the lower back (McGill, 2002). That is the reason for selecting this specific exercise.

It has been reported that a modification to the abdominal crunch could target the rectus abdominis, and the external and internal obliques, as well as the transversus abdominis by drawing the navel down towards the floor while performing the abdominal crunch (Karst & Willett, 2004). This was part of the instructions provided to patients when stabilising. By thus drawing in the navel they could activate the obliques, as well as the transversus abdominis in one exercise instead of having to do several exercises for each muscle group. This could be an important considering factor when time and patient compliance become issues.

The patients in the control group were not actively taught to stabilise like the experimental group. They had to read it in the back school document. Only two patients from the control group requested further explanation on the specific technique. It is thus unlikely that all patients in the control group performed the navel drawing-in technique. This fact may also explain the lack of improvement in the control group for the lateral musculature besides not performing any specific exercises of the lateral musculature. The experimental group performed the abdominal crunch with the naval drawing-in technique and could thus increase the amount of strengthening in the lateral musculature tests.

**Statistically significant differences were found at the 5% level of significance between the following pre-test and post-test variables within the experimental group:**

- Sixty Degree Flexor: The pre-test measurement was significantly lower than the post-test measurement in the experimental group.

This result was expected due to the nature of the design of the experimental group exercise programme. One exercise was used throughout the programme with the only difference being that it was progressed in terms of difficulty by adding an unstable surface and/or adding more repetitions. The test itself has been shown to be relatively safe for those with chronic low back pain (McGill, 2002) and no patients reported that they felt discomfort and/or pain after completion of the test.

Comparing the test and the exercise might be considered a weakness of the present study due to the fact that the test itself is an isometric test emphasising time and the exercise was a dynamic movement that emphasised repetitions performed. It has been shown that the rectus abdominis flexes the trunk (McGill, 2002), but also seems to have a function of stability (Friedli *et al.*, 1988; Zattara & Bouisset, 1988; Aruin & Latash, 1995). It has also been shown to be the most active muscle during sit-ups and curl-up exercises (Juker *et al.*, 1998). This shows that the exercise selected was the correct one for the goal in mind. Here it might have been more appropriate to have selected a dynamic test to compare with dynamic movements, or to have selected an isometric test to compare with an isometric exercise. However,

there was a significant improvement in the result of this test, suggesting that the isometric-dynamic combination of testing and exercise still show significant improvements in pain and disability, as shown by the pain and disability results.

Also, patients in the experimental group were instructed to perform the abdominal exercise with lumbar stabilisation. This had the added effect of stabilising the pelvis, thus promoting lumbopelvic neuromuscular control, which maintained the neutral curve in the low back (Perkins & Zipple, 2003).

**There was no significant difference between the pre-test and post-test values in the control group for this variable.**

This result was expected due to the fact that the control group programme contained no specific exercise to increase abdominal strength. This might be considered a weakness of the present study. The importance of the abdominal muscles has been discussed previously and specific but safe exercises have to be included in any back rehabilitation programme. There was a degree of strengthening involved, but this could be the consequence of other exercises requiring abdominal activation and thus strengthening the abdominal muscles.

**There was no significant difference between the control and experimental groups at post-test for this variable.**

This again showed the effectiveness of the experimental exercise programme. It improved more than the control group for this value, though not significantly.

**No statistically significant difference was found between any of the other post-test measurements between the experimental and control groups.**

It can therefore be concluded that, as far as the post-test measurements are concerned, the two groups were very similar regarding the latter variables. This result only partially confirms the hypothesis of the study, which stated that the aggressive-progressive exercise programme would lead to more of an improvement in low back pain variables than a more conservative exercise programme.



The results were very similar for the two programmes. Both groups improved significantly, which was a good result for the study, but it was hoped that the aggressive-progressive group would improve more. More aggressive types of exercise programmes have been used for low back pain in the past (Manniche *et al.*, 1991; Johannsen *et al.*, 1995; Hartigan *et al.*, 2000; Petersen *et al.*, 2002). Results have shown that pain and disability decrease, and functionality increases with more intensive exercise programmes (Hartigan *et al.*, 2000).

It has been argued that it is more important to focus on the consequences of pain rather than focusing only on pain treatment (Staal *et al.*, 2005). This argues in favour of more aggressive exercise treatments, which will help to improve functional status because it will include more exercises than merely just stabilisation exercises. More aggressive types of exercise programmes will not necessarily be more harmful to the low back (Videman *et al.*, 1995) if the programme is designed sensibly and scientifically.

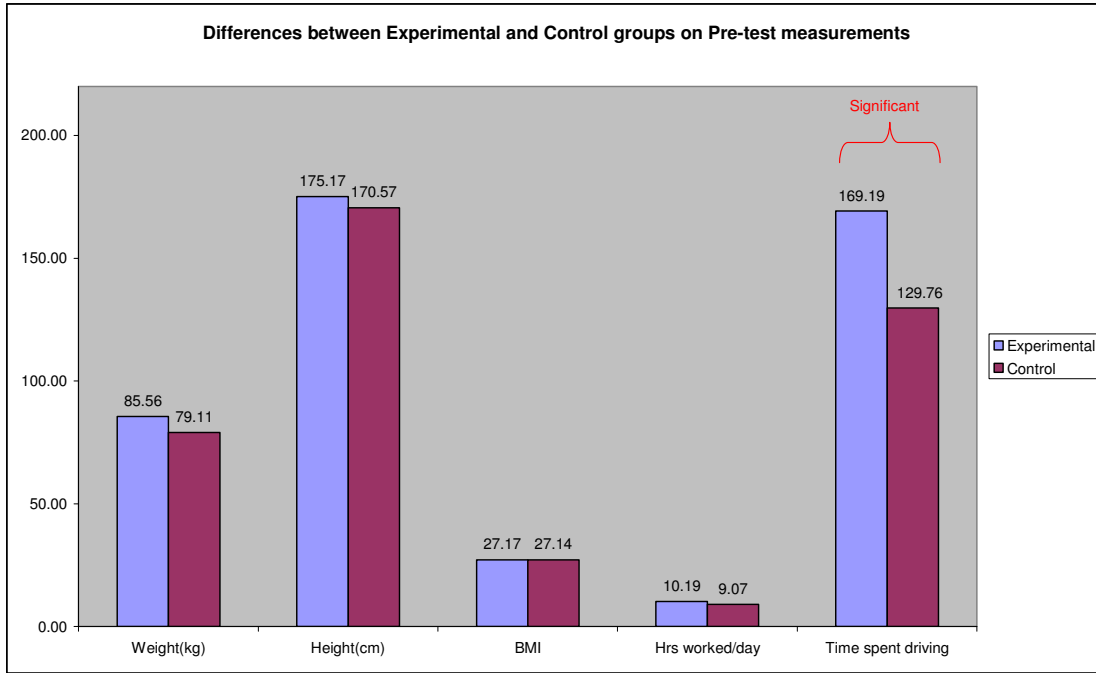
**Table 4.10: Neuropathic Pain Results**

	<b>N</b>	<b>Mean</b>
<b>Neuropathic Pain Score (DN4)</b>	15	1.4

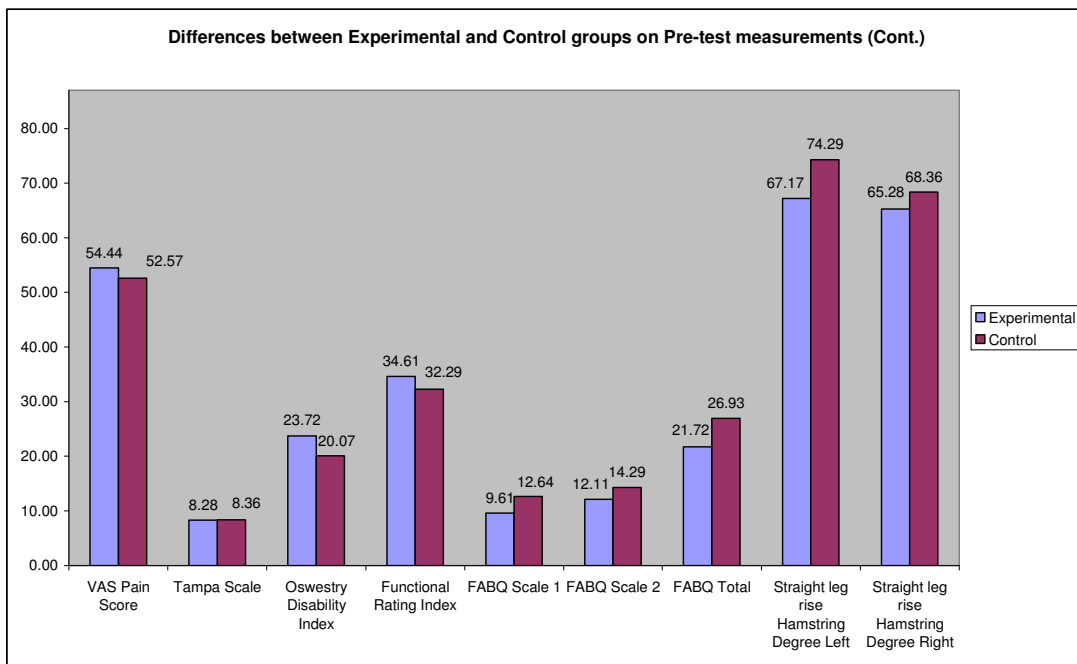
According to the DN4 questionnaire a score of 4 and higher is a positive indication of neuropathic pain. The mean score was only 1.4, indicating that the sample did not have a neuropathic pain component. However, the important implication that neuropathic pain has, has been realised in recent years (Meyer, 2007). More research is needed on this topic, especially with regard to exercise and a possible treatment option for exercise.

#### **4.5.3 Results of Differences Between Experimental and Control Groups on Pre-test Measurements**

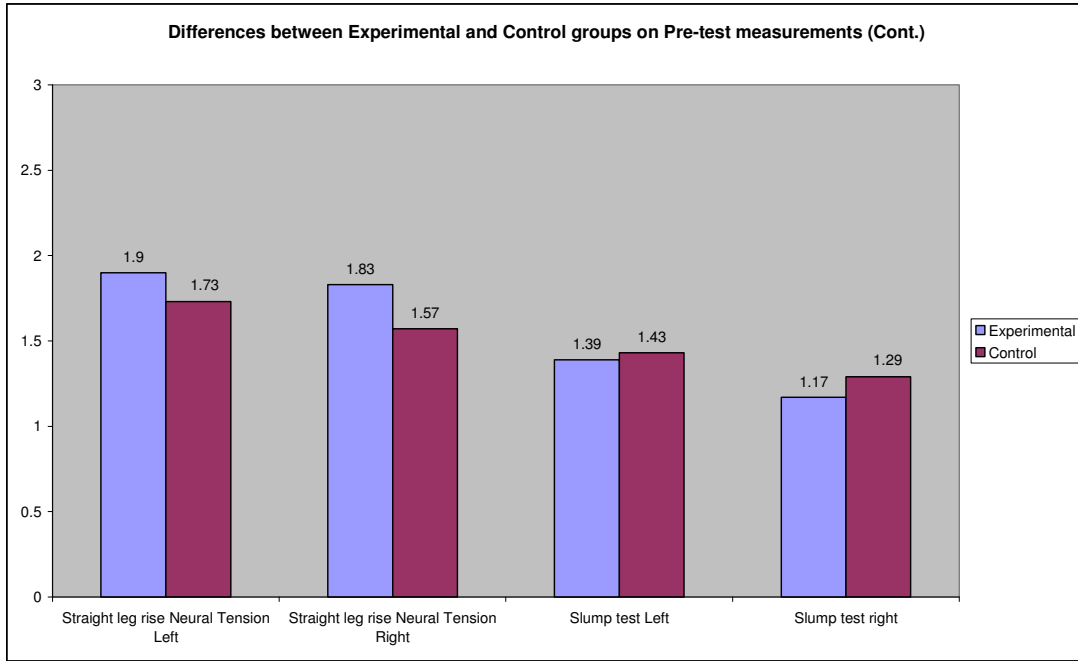
Results are summarised in Figure 4.1 to 4.4.



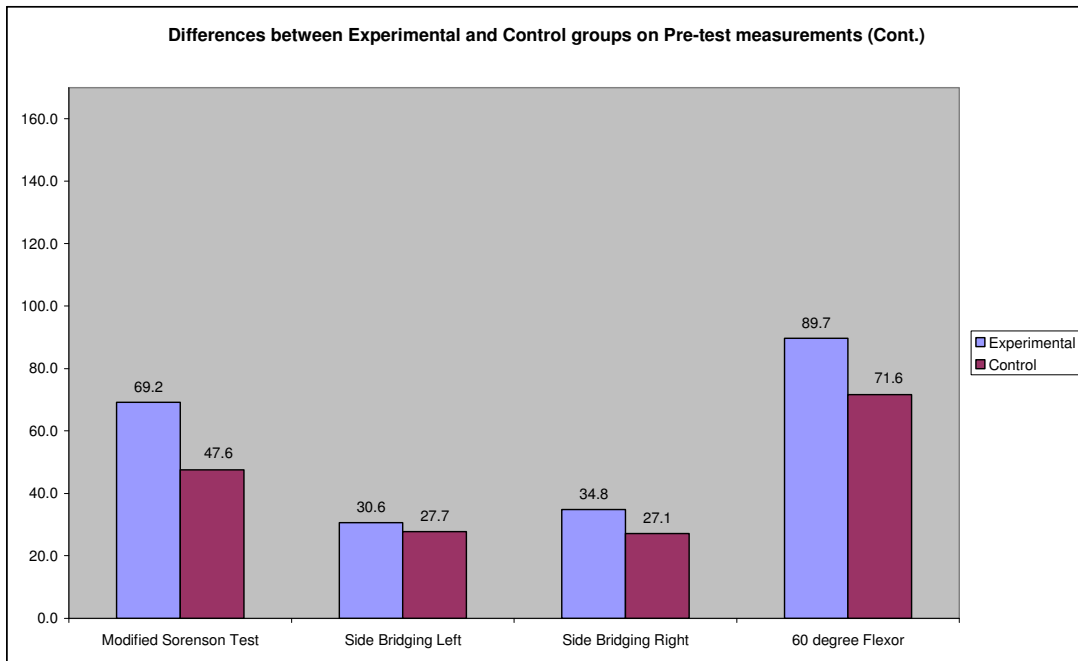
**Figure 4.1: Difference Between Experimental and Control Groups on Pre-test Measurements**



**Figure 4.2: Difference Between Experimental and Control Groups on Pre-test Measurements (continued)**



**Figure 4.3: Difference Between Experimental and Control Groups on Pre-test Measurements (continued)**



**Figure 4.4: Difference Between Experimental and Control Groups on Pre-test Measurements (continued)**

#### 4.5.4. Results of Differences Between Experimental and Control Groups on Post-test Measurements

Statistically significant differences at the 5% level of significance of the following were found between the post-test measurements of the experimental and control groups:

- Straight Leg Rise Hamstring Degree Right: The post-test value of the experimental group was significantly higher than that of the control group.
- Side Bridging Left: The post-test value of the experimental group was significantly higher than that of the control group.
- Side Bridging Right: The post-test value of the experimental group was significantly higher than that of the control group.

No statistically significant difference was found between any of the other post-test measurements between the experimental and control groups. It can therefore be concluded that, as far as the post-test measurements are concerned, the two groups were very similar for the latter variables. Results are summarised in Figures 4.5 to 4.7 that follow.

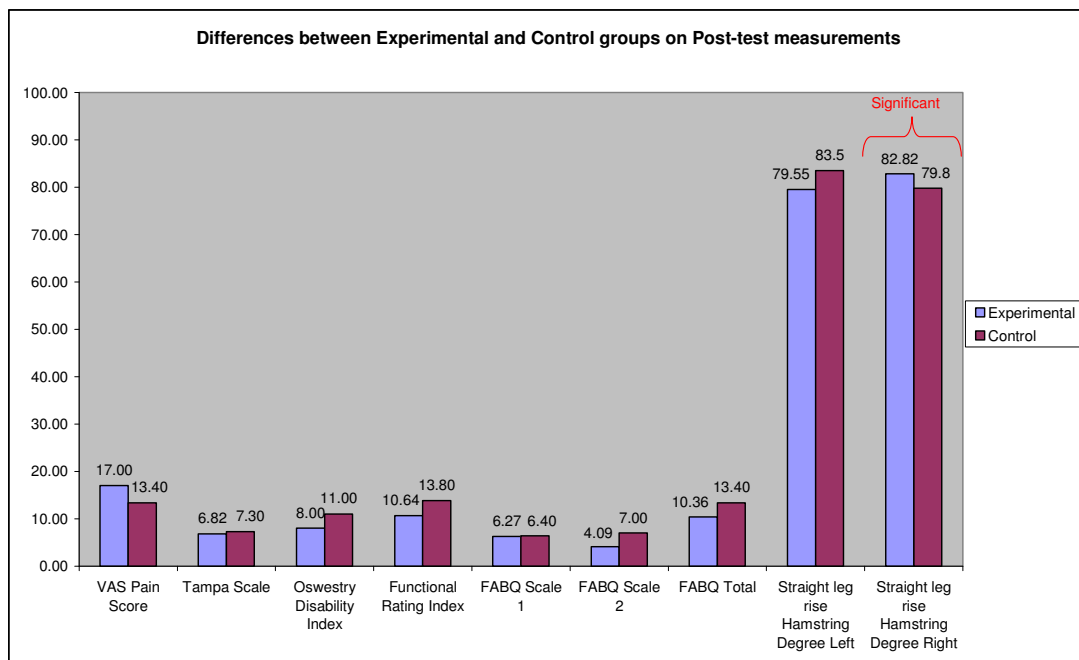
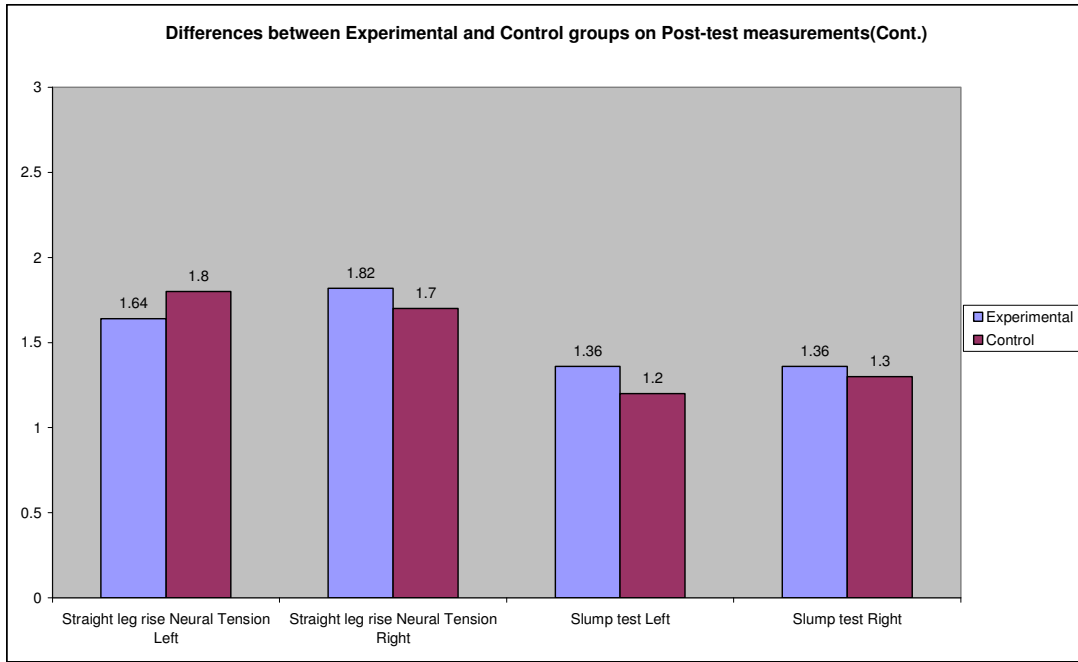
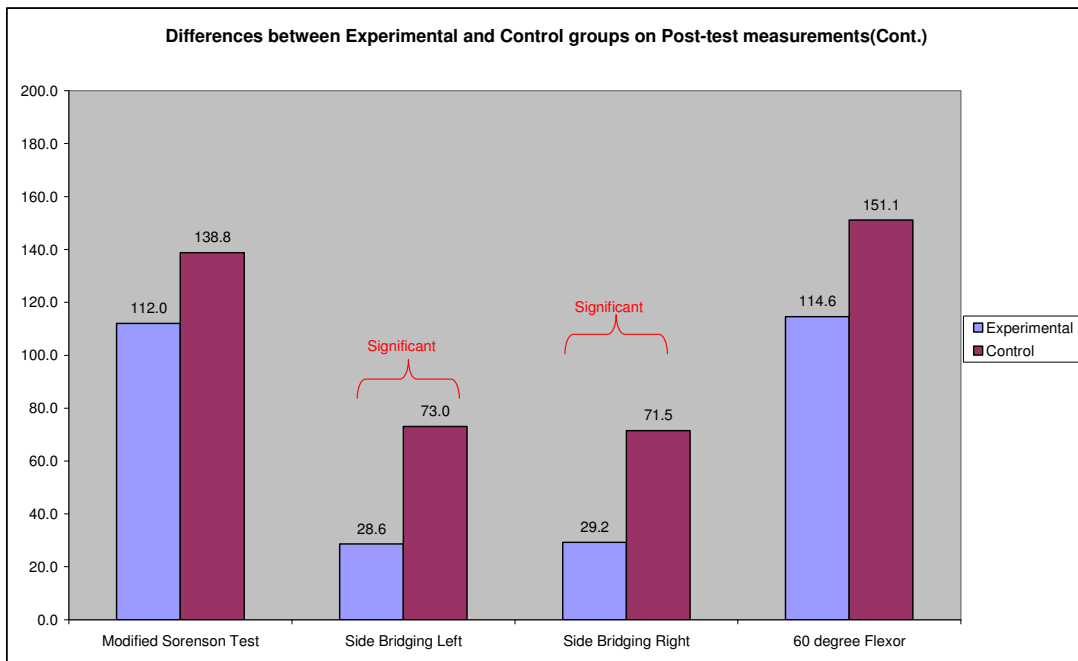


Figure 4.5: Difference Between Experimental and Control Groups on Post-test Measurements



**Figure 4.6: Difference Between Experimental and Control Groups on Post-test Measurements (continued)**



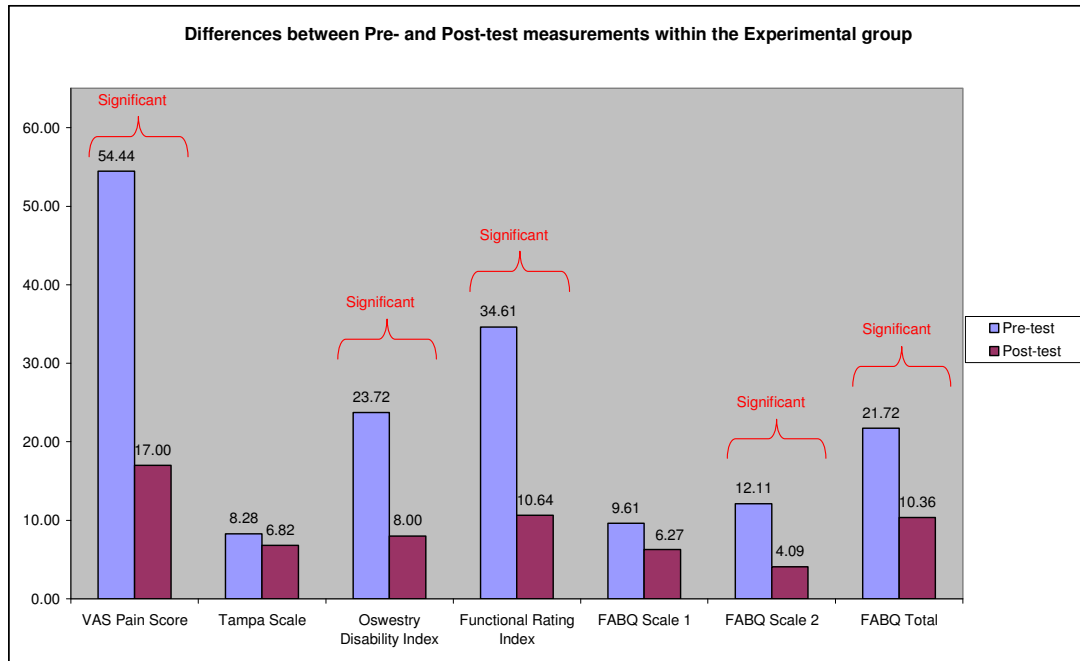
**Figure 4.7: Difference Between Experimental and Control Groups on Post-test Measurements (continued)**

#### **4.5.5 Results of the Analysis to Test Whether Statistically Significant Differences Existed Between the Pre-test and Post-test Measurements Within the Experimental Group**

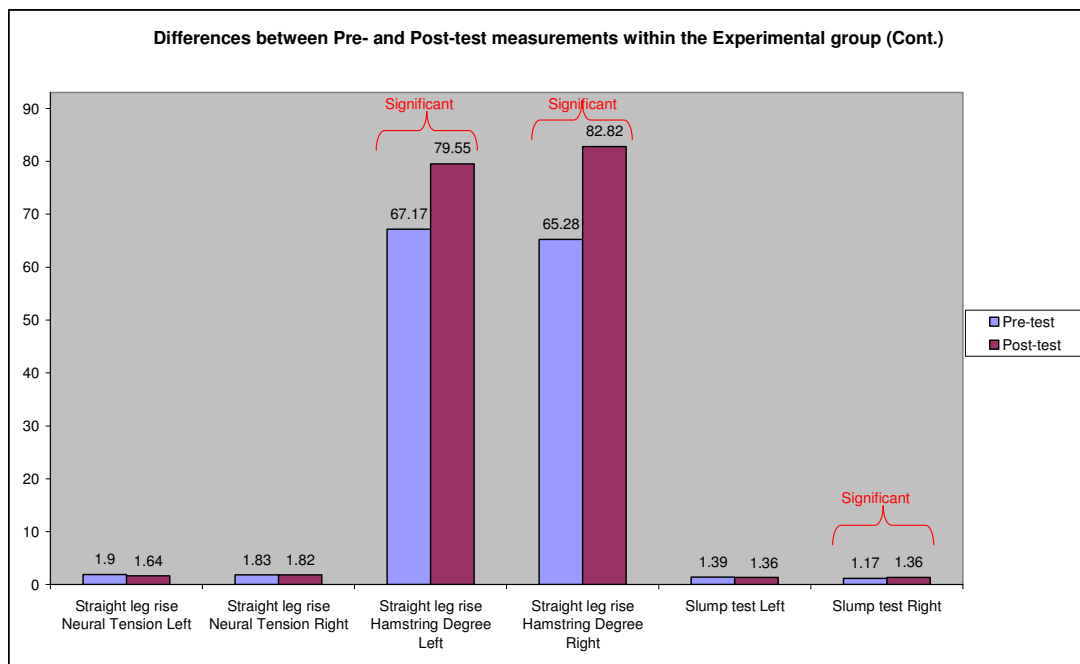
**Statistically significant differences were found at the 5% level of significance between the following pre-test and post-test variables within the experimental group:**

- VAS Pain Score: The pre-test measurement was significantly higher than the post-test measurement.
- Oswestry Disability Index: The pre-test measurement was significantly higher than the post-test measurement.
- Functional Rating Index: The pre-test measurement was significantly higher than the post-test measurement.
- FABQ Scale 2: The pre-test measurement was significantly higher than the post-test measurement.
- FABQ Total: The pre-test measurement was significantly higher than the post-test measurement.
- Straight Leg Rise Hamstring Degree Left: The post-test measurement was significantly higher than the pre-test measurement.
- Straight Leg Rise Hamstring Degree Right: The post-test measurement was significantly higher than the pre-test measurement.
- Slump Test Right: The post-test measurement was significantly higher than the pre-test measurement.
- Modified Sorenson Test: The pre-test measurement was significantly lower than the post-test measurement in the experimental group.
- Side Bridging Left: The post-test measurement was significantly higher than the pre-test measurement in the experimental group.
- Side Bridging Right: The pre-test measurement was significantly lower than the post-test measurement in the experimental group.
- 60 Degree Flexor: The pre-test measurement was significantly lower than the post-test measurement in the experimental group.

No statistically significant differences at the 5% level of significance were found between any of the other pre-test and post-test measurements within the experimental group. Results are summarised in figures 4.8 to 4.10 that follow.

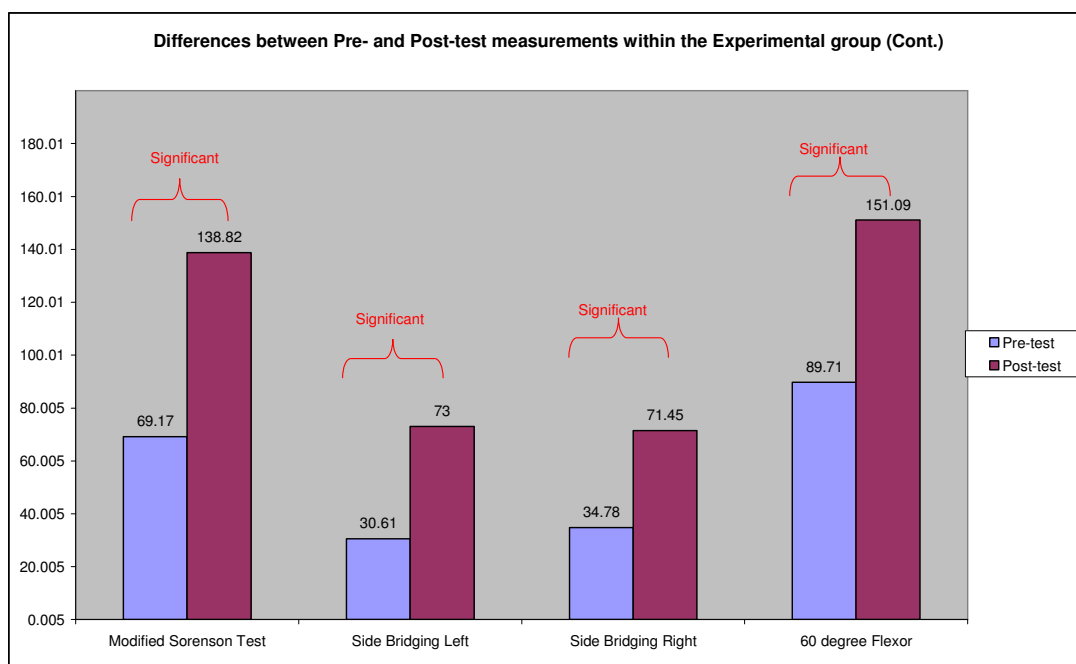


**Figure 4.8: Difference Between Pre-test and Post-test Measurements within the Experimental Group**



**Figure 4.9: Difference Between Pre-test and Post-test Measurements within the Experimental Group (continued)**





**Figure 4.10: Difference Between Pre-test and Post-test Measurements within the Experimental Group (continued)**

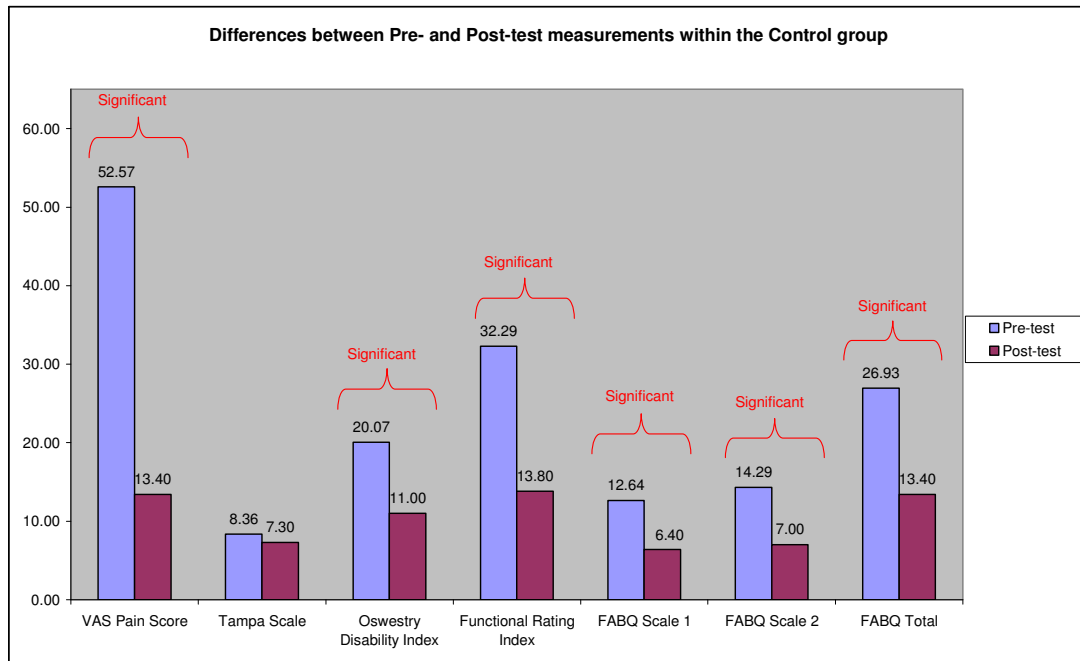
#### 4.5.6 Results of the Analysis to Test Whether Statistically Significant Differences Existed Between the Pre-test and Post-test Measurements Within the Control Group

Statistically significant differences were found at the 5% level of significance between the following pre- and post-test variables within the control group:

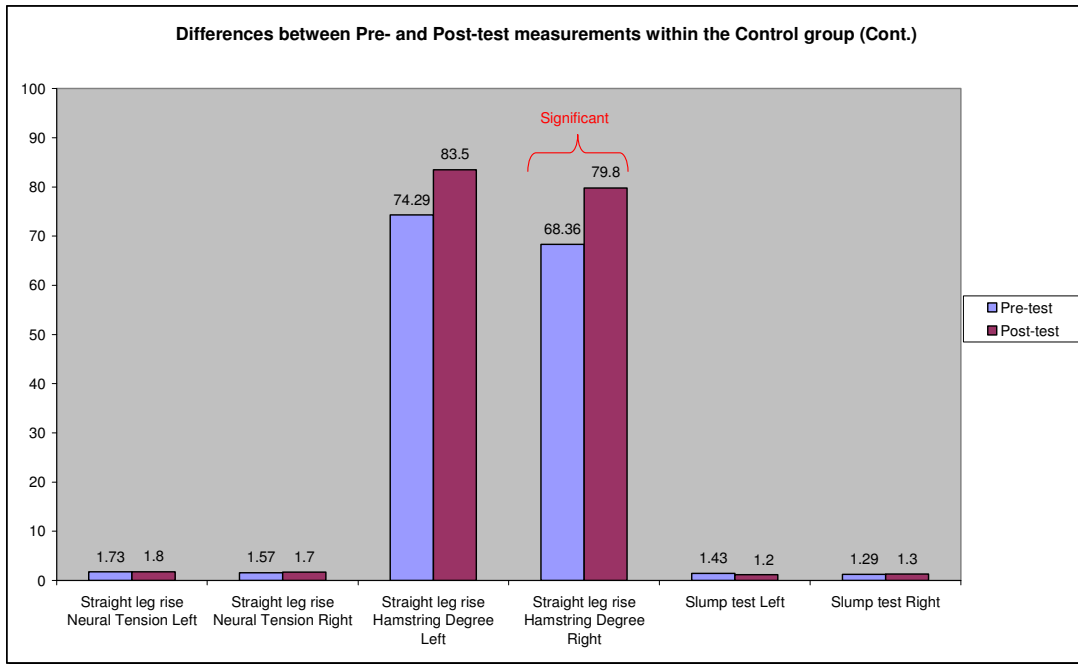
- VAS Pain Score: The pre-test measurement was significantly higher than the post-test measurement.
- Oswestry Disability Index: The pre-test measurement was significantly higher than the post-test measurement.
- Functional Rating Index: The pre-test measurement was significantly higher than the post-test measurement.
- FABQ Scale 1: The pre-test measurement was significantly higher than the post-test measurement.
- FABQ Scale 2: The pre-test measurement was significantly higher than the post-test measurement.

- FABQ Total: The pre-test measurement was significantly higher than the post-test measurement.
- Straight Leg Rise Hamstring Degree Right: The post-test measurement was significantly higher than the pre-test measurement.
- Modified Sorenson Test: The pre-test measurement was significantly lower than the post-test measurement in the control group.

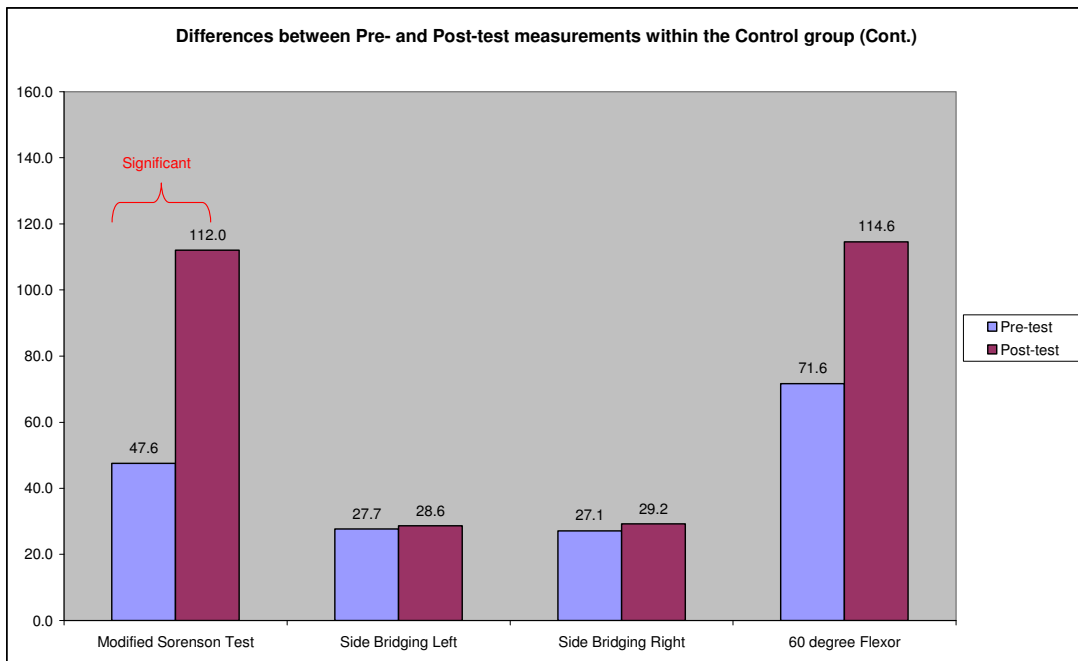
No statistically significant differences at the 5% level of significance were found between any of the other pre-test and post-test measurements within the control group. Results are summarised in figures 4.11 to 4.13 that follow.



**Figure 4.11: Difference Between Pre-test and Post-test Measurements within the Control group**



**Figure 4.12: Difference Between Pre-test and Post-test Measurements within the Control Group (continued)**



**Figure 4.13: Difference Between Pre-test and Post-test Measurements within the Control Group (continued)**

## 4.6 Case Studies

The present study utilised a 12 week, twice a week training (24 sessions) and back school regime. The ideal scenario would be a continual 24 sessions without missing a single session. However, due to the nature of the patients being full working capacity adults, this situation was not possible. Not one patient was able to complete a continual 24 sessions. Reasons include disease like colds and flu, working and family responsibility. However, most patients completed the programme within one or two weeks after the protocol was supposed to end. Some took much longer, as much as a month extra, to complete the programme.

The case studies will focus on two patients: one who completed the programme very close to 12 weeks and another who completed the programme in 16 weeks. Results on which will be focused, include important variables identified in the present study, such as pain (measured by the VAS pain scale), disability (measured by the Oswestry Disability Index and Functional Rating Index) and back extensor endurance (measured by the modified Sorensen test).

### 4.6.1 Patient A

Patient A is a 20-year-old female with a height of 1.73 m and a weight of 110 kg. Her history includes a diagnosis of mechanical back pain by a general practitioner. This has lasted for a year to date. She was advised to take part in an exercise programme for her low back pain. She reports working for 12 hours per day (student at university and part-time work for extra income). She reports high influence of back pain on her daily activities but also reports a need to have to continue with daily activities regardless of back pain. She was randomly allocated to the experimental group.

#### 4.6.1.1 Pre-test Results

Her results at pre-test were as follows: The VAS score was measured at 54. Her Oswestry Disability Index score was 18 and her Functional Rating Index score was 20, while her back extension score was 0:58 minutes, with pain reported the reason for terminating the test. The mean VAS score in the experimental group at pre-test was 54.44; the Oswestry score was 23.72, the Functional Rating Index score was

34.61 and the back extension mean score was 69.17 seconds. The comparative scores were as follows:

**Table 4.11: Mean Scores of Experimental group Compared to Patient A at Pre-test**

Variable	Mean Score	Patient A Score
VAS Pain Score	54.44	54
Oswestry Disability Score	23.72	18
Functional Rating Score	34.61	20
Back Extension Score	1:10 minutes	0.58 minutes

The scores of Patient A were very close to those of the entire group. Her disability scores were slightly lower than the mean score. Patient A was able to complete the total programme in 12.5 weeks, which was the highest completion rate of all patients.

#### 4.6.1.2 Post-test Results

Her VAS score improved to score 0 at post-test. She reported no pain at all at post-test. Her Oswestry Disability Index score and Functional Rating Index score also improved to 0. She reported that she experienced no disability at all at post-test. Her Sorensen back extension test score also improved, which measured at 3:00 minutes at post-test. She reported no problems during the test. The mean score of the group was measured at 17 for the VAS pain score, 8 for the Oswestry Disability Index, 10.64 for the Functional Rating Index and 2.3 minutes for the back extension test. The comparative scores are shown in the table below.

**Table 4.12 : Mean Scores of Experimental Group Compared to Patient A at Post-test**

Variable	Mean Score	Patient A Score
VAS Pain Score	17	0
Oswestry Disability Score	8	0
Functional Rating Score	10.64	0
Back Extension Score	2.3 minutes	3:00 minutes

As can be seen from the post-test scores, Patient A improved clinically in comparison to the mean score of the group. It can thus be argued that her scores improved due to her regular attendance of sessions and commitment to the programme.

Refer to Annexure D for patient report at end of the study.

#### 4.6.2 Patient B

Patient B is a 22-year-old female with a height of 1.60 m and a weight of 59 kg. Her history includes a 4-year period of continuous pain. She had been participating in modern dancing activities for 17 years, which was identified as a possible causative factor in her back pain. She also has poor posture. She has been advised by her general practitioner to participate in a rehabilitation programme for her low back pain. She reports working for 16 hours per day (student at university and part-time work for extra income, as well as teaching dance classes). She reports high influence of back pain on her daily activities but also reports a need to have to continue with daily activities regardless of back pain. She was randomly allocated to the control group.

##### 4.6.2.1 Pre-test Results

Her results at pre-test were as follows: The VAS score was measured at 59. Her Oswestry Disability Index score was 10 and her Functional Rating Index score was 23, while her back extension score was 0:32 minutes, with pain and weakness being reported as the reasons for terminating the test. The mean VAS score in the control group at pre-test was 52.57, the Oswestry score was 20.07, the Functional Rating Index score was 32.29 and the back extension mean score was 47.57 seconds. The comparative scores are shown in Table 4.13.

**Table 4.13: Mean Scores of Control Group Compared to Patient B at Pre-test**

Variable	Mean Score	Patient B Score
VAS Pain Score	52.57	59
Oswestry Disability Score	20.07	10
Functional Rating Score	32.29	23
Back Extension Score	0:47 minutes	0:32 minutes

The scores of Patient B were very different to those of the control group mean score. Her disability score was much lower than that of the mean score. She reported that she was still able to function despite her long history of low back pain. Patient B was able to complete the total programme in 16 weeks, which was the worst completion rate of all patients.

#### 4.6.2.2 Post-test Results

Her VAS score improved to score 11 at post-test. She reported minimal pain and discomfort at post-test. Her Oswestry Disability Index score was measured at 2 and her Functional Rating Index score was measured at 5. She reported that she experienced no disability at all at post-test. Her Sorensen Back Extension test score did not show much improvement, being measured at 0:37 minutes at post-test. She reported anticipation of pain as the reason for terminating the test. The mean score of the group was measured at 13.40 for the VAS pain score, 11.00 for the Oswestry Disability Index, 13.80 for the Functional Rating Index and 1.9 minutes for the Back Extension test. The comparative scores are shown in Table 4.14 below.

**Table 4.14 : Mean Scores of Control Group Compared to Patient B at Post-test**

Variable	Mean Score	Patient B Score
VAS Pain Score	13.40	11
Oswestry Disability Score	11	2
Functional Rating Score	13.80	5
Back Extension Score	1.9 minutes	0:37 minutes

As can be seen from the post-test scores, Patient B improved clinically in comparison to the mean scores of the group. Pain levels improved slightly, but there were better improvements in the disability scores. Her back extension score was much weaker than the mean score of the group and only slightly better than her pre-test score.

Her improvement from pre-test to post-test was clinically relevant. This can be explained by two possible reasons: Firstly, because her attendance was irregular, it



could be argued that her body did not receive the exercise stimulus regularly enough, and the improvement was not as great as some of the other patients in the control group who attended regularly. Secondly, because she has a much longer history of low back pain, her pain became very chronic. The effects of chronic pain had a longer time to develop in her than in some other patients. The effects of chronic pain had a longer time to develop in her than in some other patients.

Refer to Annexure D for patient report at end of the study.

**Table 4.15: Comparison of Post-Test scores for Patient A and Patient B**

<b>Variable</b>	<b>Patient A Score</b>	<b>Patient B Score</b>
VAS Pain Score	0	11
Oswestry Disability Score	0	2
Functional Rating Score	0	5
Back Extension Score	3:00 minutes	0:37 minutes

As shown in the Table 4.15 above, both patients' scores improved clinically. Only Patient B's back extension test score was poor, but the rest of the scores showed clinical improvement. From this table, it is clear that the exercise score of the regular attending patient (Patient A) was clinically better than that of the irregular attendant (Patient B). This provides evidence of the importance of regular attendance of training sessions concerning low back pain. Exercise stimulus needs to be provided regularly to enable the body to adapt to the training stimulus and to thus improve (Ahtiainen & Häkkinen, 2009; Hawley, 2009). Exercise cannot be completed irregularly and expected to have improved results in the physical testing.

## CHAPTER 5

### SUMMARY, CONCLUSION AND RECOMMENDATIONS

#### 5.1 Introduction

The purpose of this study was to determine if an aggressive-progressive training programme would be more effective than more traditional conservative training programmes on subjects with chronic low back pain. Test variables included self-reported pain, kinesiphobia, disability, fear avoidance behaviour, rate of perceived exertion, time spent working and driving, neuropathic pain and several physical variables, which included low back extension endurance, lateral trunk muscle endurance, abdominal flexor endurance and demographic data such as age, height, weight and body mass index (BMI).

The subjects were randomised into a conservative exercising group and an aggressive-progressive exercise group. The conservative exercise group was labelled as the control group and the experimental [aggressive-progressive] group was labelled as the experimental group. Both groups trained twice per week under supervision for 12 weeks. After the 12 week intervention period all of the data was collected again and compared with the pre-test data.

#### 5.2 Summary of Results

Main findings will be summarised in the following section.

- A statistically significant difference was found at the 5% level of significance between the experimental and control group for pre-test transport/driving time. The transport/driving time for the experimental group was significantly higher than that of the control group. No statistically significant differences were found between the pre-test of the experimental and control group for any of the other measurements. It can thus be concluded that, as far as the rest of the pre-test measurements are concerned, the two groups were very similar.

- Statistically significant differences at the 5% level of significance were found between the post-test measurements of the experimental and control group of the following:
  - Straight Leg Rise Hamstring Degree Right: The post-test value of the experimental group was significantly higher than that of the control group.
  - Side Bridging Left: The post-test value of the experimental group was significantly higher than that of the control group.
  - Side Bridging Right: The post-test value of the experimental group was significantly higher than that of the control group.
  
- No statistically significant difference was found between any of the other post-test measurements between the experimental and control group. It can therefore be concluded that, as far as the post-test measurements are concerned, the two groups were very similar with respect to the latter variables.
  
- Statistically significant differences were found at the 5% level of significance between the following pre-test and post-test variables within the experimental group:
  - VAS pain score: The pre-test measurement was significantly higher than the post-test measurement.
  - Oswestry Disability Index: The pre-test measurement was significantly higher than the post-test measurement.
  - Functional Rating Index: The pre-test measurement was significantly higher than the post-test measurement.
  - FABQ Scale 2: The pre-test measurement was significantly higher than the post-test measurement.
  - FABQ total: The pre-test measurement was significantly higher than the post-test measurement.
  - Straight Leg Rise Hamstring Degree Left: The post-test measurement was significantly higher than the pre-test measurement.
  - Straight Leg Rise Hamstring Degree Right: The post-test measurement

- was significantly higher than the pre-test measurement.
- Slump Test Right: The post-test measurement was significantly higher than the pre-test measurement.
  - Modified Sorenson Test: The pre-test measurement was significantly lower than the post-test measurement in the experimental group.
  - Side Bridging Left: The post-test measurement was significantly higher than the pre-test measurement in the experimental group.
  - Side Bridging Right: The pre-test measurement was significantly lower than the post-test measurement in the experimental group.
  - 60 Degree Flexor: The pre-test measurement was significantly lower than the post-test measurement in the experimental group.
- No statistically significant differences at the 5% level of significance were found between any of the other pre-test and post-test measurements within the experimental group.
  - Statistically significant differences were found at the 5% level of significance between the following pre-test and post-test variables within the experimental group:
    - VAS pain score: The pre-test measurement was significantly higher than the post-test measurement.
    - Oswestry Disability Index: The pre-test measurement was significantly higher than the post-test measurement.
    - Functional Rating Index: The pre-test measurement was significantly higher than the post-test measurement.
    - FABQ Scale 1: The pre-test measurement was significantly higher than the post-test measurement.
    - FABQ Scale 2: The pre-test measurement was significantly higher than the post-test measurement.
    - FABQ total: The pre-test measurement was significantly higher than the post-test measurement.
    - Straight Leg Rise Hamstring Degree Right: The post-test measurement was significantly higher than the pre-test measurement.

- Modified Sorenson Test: The pre-test measurement was significantly lower than the post-test measurement in the control group.
- No statistically significant differences at the 5% level of significance were found between any of the other pre-test and post-test measurements within the control group.

### 5.3 Conclusion

Chronic low back pain does have a high prevalence rate with up to 84% reported in developed countries (Walker, 2000; Simmonds & Dreisinger, 2003). It constitutes a very high economic and personal cost from both medical usage, and loss of work time and production (Rizzo *et al.*, 1998; Childs *et al.*, 2004; Luo *et al.*, 2004; Louw *et al.*, 2007). This may lead to disability, which may increase the burden of low back pain if it occurs at an early age (Punnett *et al.*, 2005). Focus has to be to return those with chronic low back pain to work as soon as possible.

The low back has been recognised as a structure with intricate functions to maintain stability, which is crucial for safe and effective movement (Panjabi, 1992; Granata & England, 2006). The passive stabilising structure (bone, ligaments and discs) is the first part of this low back structure, and it has to absorb pressure without being able to change its' composition. The neural system forms the second component of the low back structure, and has to be able to make adaptations to changing stresses. The muscle system is the third part of the low back structure, and this part forms the dynamic stabilising structure of the spine (Panjabi, 1992; Granata & England, 2006).

The muscular system consists of the global muscle system consisting of large, torque-producing muscles that act on the trunk and spine without being directly attached to it (Bergmark, 1989a). It includes the rectus abdominis, external oblique and the thoracic part of the lumbar iliocostalis. The local muscle system consists of muscles that directly attach to the lumbar vertebrae. It is responsible for providing segmental stability and directly controls the lumbar segments. By definition the multifidus, transversus abdominis and the posterior fibres of the internal oblique all form part of this local muscle system (Bergmark, 1989a). Both these systems work together to achieve total stability of the spine. It is these systems on which are

focused when performing exercises for low back pain. Stability of the spine is further achieved by performing spinal stabilisation, which ensures that the spine maintains its position of neutral spine (Lam *et al.*, 1989).

Exercise therapy has been reported to be successful in the treatment of chronic low back pain (Spitzer *et al.*, 1987; Koes *et al.*, 1991; Nordin & Campello, 1999; Van der Velde & Mierau, 2000; Albright, 2001; Friedrich *et al.*, 2005; Hayden *et al.*, 2005; Krismer & Van Tulder, 2007). Aerobic exercise alone has shown positive results, but no significant improvement compared to other types of exercise. Any exercise that increases functionality and gives the subject a feeling of control is effective in treating chronic low back pain (Petersen *et al.*, 2002). Stabilisation exercises have been shown to have the most significant results (Van Vliet & Heneghan, 2006; Tsao & Hodges, 2008). Exercise programmes that contain functional exercises, which involve both the local and global musculature combined with cognitive intervention have been shown to generate even better results (Bergmark, 1989a).

The results from the present study indicate that an aggressive-progressive exercise programme may be more effective than more conservative exercises in the treatment of chronic low back pain. Both types of programmes have shown to be very effective in the treatment of chronic low back pain in the present study, as well as in the literature (Koes *et al.*, 1991; Nordin & Campello, 1999; Van der Velde & Mierau, 2000; Friedrich *et al.*, 2005). However, more aggressive types of training programmes are suggested in the literature for the treatment of chronic low back pain (Manniche *et al.*, 1991; Johannsen *et al.*, 1995; Oldervoll, 2001; Petersen *et al.*, 2002; Ostelo *et al.*, 2003) due to the need to improve overall functionality and decrease disuse that results from the consequence of pain. Pain itself is not regarded as the limiting factor in chronic low back pain cases (Staal *et al.*, 2003; Staal *et al.*, 2005).

Because of the importance of pain being recognised as a disease in itself (Meyer, 2007) more focus needs to be placed on pain management, especially chronic pain and pain 'diseases' such as neuropathic pain. Results from the present study confirm this view. All of the subjects who volunteered for the study, even those who fell out, did so because they experienced low back pain. None wanted to participate because

they felt that their back muscles were weak or that they were suffering from fear avoidance behaviour. All of them wanted a potential cure for their low back pain. This shows a potential conflict in the expectations of subjects and the goal of research. Research recommends that the consequences of pain be addressed before pain itself is addressed, because pain is a very subjective experience. However, pain relief is the primary goal for subjects, as many of them feel that they can resume normal activities once their pain has subsided. But this illustrates unrealistic goals from subjects, as they expect their pain to disappear completely. Unfortunately it is reported that pain has a high reoccurrence rate (Staal *et al.*, 2005) and subjects often have unrealistic beliefs about pain and injury (Waddell *et al.*, 1993; Picavet *et al.*, 2002). That is why it is important to merge the goals of treatment with those of the subjects. If not, the treatment will be ineffective or the subjects will lose interest. This has to be explained to the subject (Meyer, 2007) in detail right from the start.

The experience of chronic pain has certain physical and psychological consequences. These include disability, fear avoidance behaviour, physical deconditioning and weakening of muscles, and possible permanent absence from work. All of these factors have to be addressed in order for the subject to have a favourable outcome as these factors pose a greater problem than pain alone. This can best be achieved by a multidisciplinary/interdisciplinary team consisting of various medical professionals who all work together to achieve maximum levels of success with a subject.

The present study found that a well-structured progressive-aggressive programme could be as effective as conservative exercise programmes that have been used in the past. In both groups pain levels, levels of disability and muscle endurance strength all improved. In most cases the progressive-aggressive group showed better improvements than the conservative group, although not significant. The progressive-aggressive group addressed the functional capacity component, which the conservative group did not.

The two case studies also showed significant improvements compared to the mean scores of the rest of the group. Both subjects improved more than or just as much as the group overall. In comparison they also showed good improvement, with subject A

performing better in most of the post-tests. Subject A was also the more regular attendee and finished the course only slightly overdue, while it took subject B a month longer to complete the course. This could be a reason why subject A performed better in the post-test.

These case studies provided evidence of the perception of the two subjects that none of the other tests would have been able to provide. It provided evidence of the importance for the subjects to reduce their back pain and to increase their functional ability. Although disability levels were low in the entire population group subjects were still affected by their back pain in that they had lost interest, productivity and self-satisfaction in not only recreational activities but also in work-related activities. This supports the idea of the focus on pain reduction being a secondary goal and the focus on alleviating the consequence of pain being a primary goal (Staal *et al.*, 2005).

#### **5.4 Recommendations**

From the present study the following recommendations can be made:

- Aggressive progressive exercise programmes for chronic low back pain can be very effective. The exercise programme has to contain both stabilising exercises and exercises for larger muscle groups to restore functional capacity.
- Exercise programmes should contain exercises for both the global and local muscle groups. Muscles involved in stabilisation, extension, flexion, lateral flexion and rotation should all receive attention. Research has advocated the importance of the extensor muscle groups and they should receive priority treatment. The other muscle groups should not be neglected though and have to be properly exercised as well.
- Due to the importance of restoring functional capacity other exercises need to be included in the exercise programme in order to train the larger movement muscle groups. Muscles such as those of the legs, chest and shoulders need to be included to strengthen them to restore the functional capacity tasks for which they are responsible.



- ➔ Exercise programmes should also contain some form of aerobic exercise. Due to the progression of chronic low back pain subjects tend to undergo deconditioning as a result of less participation in activities. Aerobic exercises will re-establish aerobic capacity and enable subjects to return to their normal activities without exertion inhibiting them. The aerobic exercises will also help to maintain their health status and prevent diseases such as diabetes from becoming problematic because of decreased levels of activity.
- ➔ Stretching exercises need to be done along with strengthening exercises. The stretching exercises are not done to necessarily improve the flexibility of the low back muscles, but to rather maintain the flexibility of the hips and legs. The gluteus muscle group and the quadriceps muscle group, as well as the hamstring muscle group need specific stretching exercises to maintain their mobility which is in danger as a result of restricted movement because of pain.
- ➔ It has been reported that subjects need up to approximately 20 supervised training sessions to achieve a significant amount of success (Sanders & Brena, 1993). The present study used 24 sessions. This achieved a fair amount of success. However, many subjects complained about the length of the programme and remarked that they would not have finished the programme if they had to pay for it. Many subjects felt improvement after 6-10 sessions. It is thus recommended that at least 10 sessions be done and then it could be decided to continue or to discharge the subject with home-based exercises. This is particularly important concerning those in full working capacity.
- ➔ Subjects should receive both supervised exercise training and unsupervised home training programmes. The unsupervised home training programmes should not be too long in duration or too complicated, but should still be performed at least once daily. Compliance is always a problem with unsupervised home exercise programmes. However, the subjects have to be informed of the importance of the home exercise programme from the start. He or she has to be willing to accept responsibility for his or her own treatment outcomes. The present study did not use any form of extra home

training in order to control the specific exercise activity of subjects, thereby increasing the internal validity of the study. The use of home exercises has been reported in the literature (Arokoski *et al.*, 2004; Sherman *et al.*, 2005) and it is advised to use them in conjunction with the normal regime of training for low back pain.

- ➔ Subjects have to be taught the stabilisation method or a variant thereof. This will place emphasis on the stabilisation muscles and prevent the use of the mobilisation muscles for the specific stabilisation exercises. It will further retrain these muscles and ensure the grooving of proper neuromuscular activation patterns.

## 5.5 Future Research

- ➔ More research has to be conducted on pain management, especially where exercise treatment modalities are concerned. Exercise appears to be a very effective treatment modality for low back pain, but it is not perfect. Combining exercise treatment with other modalities, especially cognitive behavioural treatment, seems to be the most effective. But pain needs special attention, especially if components such as central sensitisation and diseases such as fibromyalgia are involved. Exercise, combined with pharmaceutical intervention, can be more effective than executed separately. Especially combining medicine and exercise for low back pain with a fibromyalgic component needs further research. The type, intensity, frequency and duration of the involved exercise need to be better researched.
- ➔ Research also needs to focus on neuropathic pain combined with low back pain. None of the subjects in the present study showed signs of neuropathic pain, although three of them scored a 3 on the DN4 questionnaire. This would suggest that neuropathic pain did not play a role in presenting pain in the present study. However, neuropathic pain is identified as a major component in many people suffering from chronic low back pain. Research thus needs to focus on the use of exercise in treating neuropathic pain. Especially resistance exercise needs more emphasis.

- ➔ More intensive research needs to be done on the correct intensity, frequency and duration for all sub-groups of low back pain that will benefit from exercise intervention therapies. Establishing intensities for remedial exercise programmes can be difficult and specific guidelines for intensity are still needed. Frequency and duration should also be researched more regarding their ideal dosage. The reason is that the intensity, frequency and duration differ from study to study. The type of exercise also needs to be researched in order to establish which specific exercises would be effective and which could be eliminated. The present study used specific exercises selected from the literature. These exercises need to be used in future studies in the same intensity, frequency and duration as in the present study.
- ➔ Especially exercise intensity needs to be researched, as very few guidelines exist in the literature to guide intensity in exercise for chronic low back pain. The present study attempted to use the Borg rate of perceived exertion scale (RPE) to determine the intensity of the exercises in the experimental group. Future research needs to establish whether the RPE scale is an effective method for guiding low back pain rehabilitation exercises and whether it could be used to guide intensity as effectively as it guides high performance exercise intensity. The present study only used the RPE scale for the experimental group and not for the control group. It was also used only at the end. The RPE scale has great potential to measure exercise intensity but specific levels of safe intensities need to be established as guidelines for those with chronic low back pain.
- ➔ Future research also has to look at some of the more common exercises used in chronic low back pain research and practice. Exercises such as the alternative superman and all of its versions have to be compared more in-depth. Attempts should be made to establish what exercise is sensible for certain phases of the rehabilitation programme. Past research has used EMG studies in an attempt to determine the effectiveness of the frequently used remedial studies. Future research needs to standardise the use of these exercises in order to provide guidelines for their utilisation.

- ➔ Future research needs to establish whether the Ito test is an effective substitute for the Sorensen back extension endurance test for those with chronic low back pain. Some subjects find the Sorensen test intimidating. Therefore, a test that could theoretically place less strain on the low back could be very effective in testing those with chronic low back pain safely but still effectively. The test needs to be validated by using healthy subject data to establish norms, and to compare with those suffering from chronic low back pain and other forms of low back pain.
- ➔ When performing any type of extension endurance testing, research has to attempt to establish whether test termination is due to pain or to true muscular endurance. Pain is difficult to measure because of its subjective nature but establishing a difference between pain and muscular weakness in endurance testing will give a more accurate picture of those who are at a greater risk of developing chronic low back pain due to weakened back extensor muscles.
- ➔ More research needs to be done on those subjects in full working capacity. Results from the present study indicate that this specific population might not be as disabled as previously thought, although they have been suffering from chronic low back pain for a substantial amount of time. Incorporating remedial exercises into their daily routine is a barrier to participation. Research needs to focus on how to incorporate meaningful exercises into the daily routines of those working full-time as not to create the impression that the exercises are impeding on their daily routines. This type of future research thus needs to focus on compliance.
- ➔ Larger sample groups need to be used in similar studies. Although the logistic problems with this kind of study have been documented, larger sample groups will provide more statistically significant results. The results from the present study were clinically significant but larger groups will provide statistical and clinical significance. Especially using larger sample groups to rehabilitate the subjects through the whole rehabilitation process will give a more statistically significant meaning to the results.
- ➔ It also needs to be established whether those in full working capacity will benefit from more or from less back school time. Subjects from the present

study were all working full-time and attended the programme sessions at the end of a long working day. All were tired and their attention span was limited. Research needs to establish what amount of back school counselling will be effective, yet still transfer information and retain subject attention. The duration should not be so lengthy as to induce boredom in subjects.

## BIBIOGRAPHY

- ABENHAIM, L., ROSSIGNOL, M., VALAT, J.P., NORDIN, M., AVOUC, B., BLOTMAN, F., CHARLOT, J., DREISER, R.L., LEGRAND, E., ROZENBERG, S. & VAUTRAVES, P. (2000). The Role of Activity in the Therapeutic Management of Back Pain: Report of the International Paris Task Force on Back Pain. *Spine*. 25: 1S-33S.
- ADAMS, M.A. & HUTTON, W.C. (1988). Mechanics of the Intervertebral Disc. In: GHOSH, P. (Editor). **The Biology of the Intervertebral Disc**. Boca Raton, FL: CRC Press.
- ADAMS, M.A., MANNION, A.F. & DOLAN, P. (1999). Personal Risk Factors for First Time Low Back Pain. *Spine*. 24: 2497-2505.
- ADAMS, M.A. (2004). Biomechanics of Back Pain. *Acupuncture in Medicine*. 22: 178-188.
- AHMED, M., BJURHOLM, A., KREICHBERGS, A. & SCHULTZBERG, M. (1991). SP and CGRP Immunoreactive Nerve Fibres in the Rat Lumbar Spine. *Neurology & Orthopaedics*. 12: 19-28.
- AHMED, M., BJURHOLM, A., KREICHBERGS, A. & SCHULTZBERG, M (1993). Neuropeptide Y, Tyrosine Hydroxylase and Vasoactive Intestinal Polypeptide Immunoreactive Nerve Fibres in the Vertebral Bodies, Discs, Dura Mater and Spinal Ligaments of the Rat Lumbar Spine. *Spine*. 18: 268-270.
- AHTIAINEN, J.P. & HÄKKINEN, K. (2009). Strength Athletes are Capable to Produce Greater Muscle Activation and Neural Fatigue during High-Intensity Resistance Exercise than Non-Athletes. *Journal of Strength and Conditioning Research / National Strength & Conditioning Association*. 23(4): 1129-1134.

- ALBRIGHT, J. (2001). Philadelphia Panel Evidence-Based Clinical Practice Guidelines on Selected Rehabilitation Interventions for Low Back Pain. *Physical Therapy*. 81: 1641-1674.
- AL-OBAIDI, S.M., NELSON, R.M., AL-AWADHI, S. & AL-SHUWAIE, N. (2000). The Role of Anticipation and Fear of Pain in the Persistence of Avoidance Behaviour in Patients with Chronic Low Back Pain. *Spine*. 25: 1126-1131.
- AL-OBAIDI, S.M., AL-ZOABI, B., AL-SHUWAIE, N., AL-ZOABIE, N & NELSON, R.M. (2003). The Influence of Pain and Pain-Related Fear and Disability Beliefs on Walking Velocity in Chronic Low Back Pain. *International Journal of Rehabilitation Research*. 26: 101-108.
- AMERICAN COLLEGE OF SPORTS MEDICINE. (2000). **ACMS's Guidelines for Exercise Testing and Prescription**. (6<sup>th</sup> Edition). Philadelphia, PA: Lippincott, Williams & Wilkins.
- AMONOO-KUOFI, H.S. (1983). The Density of Muscle Spindles in the Medial, Intermediate and Lateral Columns of Human Intrinsic Postvertebral Muscles. *Journal of Biomechanics*. 136: 509-519.
- ANDERSSON, G.B.J. (1997). The Epidemiology of Spinal Disorders. In FRYMOYER, J.W. (Editor). **The Adult Spine: Principles and Practice**. Philadelphia, PA: Lippincott-Raven.
- ANDERSSON, G.B.J. (1999). Epidemiological Features of Chronic Low Back Pain. *Lancet*. 354: 581-585.

- AOKI, Y., AKEDA, K., AN, H., MUEHLEMAN, C., TAKAHASHI, K., MORIYA, H. & MASUDA, K. (2006). Nerve Fiber Ingrowth into Scar Tissue Formed Following Nucleus Pulposus Extrusion in the Rabbit Anular-Puncture Disc Degeneration Model: Effects of Depth of Puncture. *Spine*. 31(21): E774-E780.
- APKARIAN, A.V., SOSA, Y., SONTY, S., LEVY, R.M., HARDEN, N., PARRISH, T.B. & GITELMAN, D.R. (2004). Chronic Back Pain is Associated with Decreased Prefrontal and Thalamic Gray Matter Density. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*. 24: 104-105.
- ARNOLD, L.M. (2008). Management of Fibromyalgia and Comorbid Psychiatric Disorders. *The Journal of Clinical Psychiatry*. 69(2): 14-19.
- AROKOSKI, J.P., KANKAAPAA, M., VALTA, T., JUVONEN, I., PARTANEN, J., TAIMELA, S., LINDGREN, K.A. & AIRAKSINEN, O. (1999). Back and Hip Extensor Muscle Function during Therapeutic Exercises. *Archives of Physical Medicine and Rehabilitation*. 80: 842-850.
- AROKOSKI, J.P., VALTA, T, AIRAKSINEN, O. & KANKAANPÄÄ, M. (2001). Back and Abdominal Muscle Functional during Stabilization Exercises. *Archives of Physical Medicine and Rehabilitation*. 82: 1089-1098.
- AROKOSKI, J.P., VALTA, T., KANKAANPÄÄ, M. & AIRAKSINEN, O. (2004). Activation of Lumbar Paraspinal and Abdominal Muscles during Therapeutic Exercises in Chronic Low Back Pain Patients. *Archives of Physical Medicine and Rehabilitation*. 85: 823-832.
- ARUIN, A.S. & LATASH, M.L. (1995). Directional Specificity of Postural Muscles in Feed-Forward Postural Reactions during Fast Voluntary Arm Movements. *Experimental Brain Research*. 103: 323-332.



ASHBURN, M.A. & RICE, L. (1998). Interdisciplinary Approach to Pain Management in: **The Management of Pain**. New York: Churchill-Livingstone: p 235-248.

ASHBURN, M.A. & STAATS, P.S. (1999). Pain: Management of Chronic Pain. **Lancet**. 353(9167): 1-11.

ASHTON, I.K., ROBERTS, S., JAFFRAY, D.C., POLAK, J.M. & EISENSTEIN, S.M. (1994). Neuropeptides in the Human Intervertebral Disc. **Journal of Orthopaedic Research: Official Publication of the Orthopaedic Research Society**. 12: 186-192.

ASPDEN, R.M. (1989). The Spine as an Arch: A New Mathematical Model. **Spine**. 14: 266-274.

ASPDEN, R.M. (1992). Review of the Functional Anatomy of the Spinal Ligaments and the Lumbar Erector Spinae Muscles. **Clinical Anatomy**. 5: 372-387.

ASTIN, J.A. (2004). Mind Body Therapies for the Management of Pain. **The Clinical Journal of Pain**. 20: 27-32.

ÅSTRAND, P.O. & RODAHL, K. (1991). **Textbook of Working Physiology. Physiological Bases of Exercise**. (3<sup>rd</sup> Edition). New York, NY: McGraw-Hill.

BABBIE, E. (1992). **The Practice of Social Research**. (6<sup>th</sup> Edition). California, USA: Wadsworth Publishing Company.

BAGNALL, K.M., FORD, D.M., MCFADDEN, K.D., GREENHILL, B.J. & RASO, V.J. (1984). The Histochemical Composition of Human Vertebral Muscle. *Spine*. 9: 470-473.

BAJEK, S., BOBINAC, D., BAJEK, G., VRANIC, T.S., LAH, B. & DRAGOJEVIC, D.M. (2000). Muscle Fibre Distribution in Multifidus Muscle in Cases of Lumbar Disc Herniation. *Acta Medica Okayama*. 54(6): 235-241.

BALDWIN, M.C. (1999). Psychological and Physiological Influences of Hatha Yoga Training on Healthy, Exercising Adults. *Dissertation Abstracts International*. 60: 1031.

BANIC, B., PETERSEN-FELIX, S., ANDERSEN, O.K., RADANOV, B.P., VILLIGER, P.M., ARENDT-NIELSE, L. & CURACOLO, M. (2004). Evidence for Spinal Cord Hypersensitivity in Chronic Pain after Whiplash Injury and in Fibromyalgia. *Pain*. 107: 7-15.

BARKER, K.L., DAWES, H., HANSFORD, P. & SHAMLEY, D. (2003). Perceived and Measured Levels of Exertion of Patients with Chronic Back Pain Exercising in a Hydrotherapy Pool. *Archives of Physical Medicine and Rehabilitation*. 84: 1319-1323.

BASBAUM, A.I. & JESSELL, T.M. (1999) The Perception of Pain. In KANDEL, E.R. SCHWARTZ, J.H. & JESSELL, T.M. (Editors) **Principles of Neural Science**. McGraw-Hill, New York (4<sup>th</sup> Edition): pp 472-491.

BASMAJIAN, J. (1977). Motor Learning and Control: A Working Hypothesis. *Archives of Physical Medicine and Rehabilitation*. 58: 38-41.

BATEMAN, A., CULPAN, F.J., PICKERING, A.D., POWELL, J.H., SCOTT, O.M. & GREENWOOD, R.J. (2001). The Effect of Aerobic Training on Rehabilitation

Outcomes after Recent Severe Brain Injury: A Randomized Controlled Evaluation. ***Archives of Physical Medicine and Rehabilitation.*** 82: 174-182.

BEAUDOIN, C.M., CREWS, D.J. & MORGAN, D.W. (1998). Influence of Psychogenic Factors during a Prolonged Maximal Run. ***Journal of Sports Behaviour.*** 21: 377-386.

BEITH, I.D., SYNNOTT, R.E.&NEWMAN, S.A. (2001). Abdominal Muscle Activity during the Abdominal Hollowing Maneuver in the Four Point Kneeling and Prone Positions. ***Manual Therapy.*** 6: 82-87.

BELEN'KII, V., GURFINKEL, V.S. & PALTSEV, Y. (1967). Elements of Control of Voluntary Movements. ***Biofizika.*** 12: 135-141.

BELMONTE, C & CERVERO, E. (1996). ***Neurobiology of Nociceptors.*** Oxford, UK:OxfordUniversity Press.

BENDIX, T., BENDIX, A., LABRIOLA, M., HAESTRUP, C. & EBBEHØJ, N. (2000). Functional Restoration versus Outpatient Physical Training in Chronic Low Back Pain: A Randomized Comparative Study. ***Spine.*** 25: 2494-2500.

BENNETT, R.M. (1999). Emerging Concepts in the Neurobiology of Chronic Pain. ***Mayo Clinic Proceedings. Mayo Clinic.*** 74: 385-398.

BENTSEN, H., LINGARDE, F. & MANTHORPE, R. (1997). The Effect of Dynamic Strength Back Exercise and/or a Home Training Program in 57-Year-Old Women with Chronic Low Back Pain: Results of a Prospective Randomized Study with a 3-Year Follow-Up Period. ***Spine.*** 22: 1494-1500.

BERGMARK, A. (1989a). Stability of the Lumbar Spine: A Study in Mechanical Engineering. ***Acta Orthopaedica Scandinavica. Supplementum.*** 230: 20-24.

BERGMARK, A. (1989b). The Local and Global Systems; Instability of the Lumbar Spine. ***Acta Orthopaedica Scandinavica.*** 60: 20-24.

BERGQUIST-ULLMAN, M. & LARSSON, U. (1977). Acute Low Back Pain in Industry: A Controlled Prospective Study with Special Reference to Therapy and Confounding Factors. ***Acta Orthopaedica Scandinavica.*** 170: 1-117.

BEVAN, S. (1996). Signal Transduction in Nociceptive Afferent Neurons in Inflammatory Conditions. ***Progress in Brain Research.*** 113: 201-213.

BIEDERMANN, H.J., SHANKS, G.L., FORREST, W.J. & INGLIS, J. (1991). Power Spectrum Analysis of Electromyographic Activity. ***Spine.*** 16: 1179-1184.

BIGOS, S.J., SPENGLER, D.M., MARTIN, N.A., ZEH, J., FISHER, L. & NACHEMSON, A. (1986). Back Injuries in Industry: A Retrospective Study. III: Employee Related Factors. ***Spine.*** 3: 252: 256.

BIGOS, S., BOWYER, O. & BRAEN, G. (1994). Acute Low Back Problems in Adults. **Clinical Practice Guidelines 1994; No 14: AHCPR Publications No95-0645. Agency for Health Care Policy and Research. U.S. Department of Human Health and Human Services.**

BIERING-SORENSEN, F. (1984). Physical Measurements as Risk Indicators for Low Back Trouble over a One-Year Period. ***Spine.*** 9: 106-119.

- BILLETER, R., WEBER, H., LUTZ, H., HOWALD, H., EPPENBERGER, H.M. & JENNY, E. (1980). Myosin Types in Human Skeletal Muscle Fibres. ***Histochemistry and Cell Biology***. 65: 249-259.
- BISHOP, J.B., SZPALSKI, M., ANANTHRAMAN, S.K., MCINTYRE, D.R. & POPE, M.H. (1997). Classification of Low Back Pain from Dynamic Motion Characteristics Using an Artificial Neural Network. ***Spine***. 22: 2991-2998.
- BLOEM, B.R., ALLUM, J.H., CARPENTER, M.G. & HONEGGER, F.(2000). Is Lower Leg Proprioception Essential for Triggering Human Automatic Postural Response? ***Experimental Brain Research***. 130: 375-391.
- BOGDUK, N. (1980). A Reappraisal of the Anatomy of the Human Lumbar Erector Spinae. ***Journal of Anatomy***. 131(3): 525.
- BOGDUK, N., TYNAN, W. & WILSON, A.S. (1981). The Nerve Supply to the Human Lumbar Intervertebral Discs. ***Journal of Anatomy***. 132: 39-56.
- BOGDUK, N. & ENGEL, R., (1984). The Menisci of the Lumbar Zygapophyseal Joints: A Review of their Anatomy and Clinical Significance. ***Spine***. 9: 454.
- BOGDUK, N. & MACINTOSH, J.E. (1984). The Applied Anatomy of the Thoracolumbar Fascia. ***Spine***. 9: 164.
- BOGDUK, N. (1991). The Lumbar Disc and Low Back Pain. ***Neurosurgery Clinics of North America***. 2: 791-806.

- BOGDUK, N. & TWOMEY, L.T. (1991). **Clinical Anatomy of the Lumbar Spine.** (2<sup>nd</sup> Edition).Melbourne, AUS: Churchill Livingstone.
- BOGDUK, N., MACINTOSH, J.E. & PEARCY, M.J. (1992). A Universal Model of the Lumbar Back Muscles in the Upright Position. ***Spine.*** 17: 897-913.
- BOGDUK, N. (1997). **Clinical Anatomy of the Lumbar Spine and Sacrum.**Edinburgh, UK: Churchill Livingstone. 105.
- BOGDUK, N. (2004). Management of Chronic Low Back Pain. ***The Medical Journal of Australia.*** 180: 79-83.
- BONICA, J.J. (1953). **The Management of Pain.** Philadelphia:Lea and Febiger.
- BORG, G., LJUNGGREN, G. & CECI, R. (1985). The Increase of Perceived Exertion, Aches and Pain in the Legs, Heart Rate and Blood Lactate during Exercise on a Bicycle Ergometer. ***European Journal of Applied Physiology and Occupational Physiology.*** 54: 343-349.
- BORG,G., HASSMEN, P. & LAGERSTROM, M. (1987). Perceived Exertion Related to Heart Rate and Blood Lactate during Arm and Leg Exercise. ***European Journal of Applied Physiology.*** 65: 679-685.
- BORKAN, J.M.&CHERKIN, D.C. (1996). An Agenda for Primary Care Research on Low Back Pain. ***Spine.*** 21: 2880-2884.

- BORMS, J. & VAN ROY, P. (1996). Flexibility. In ESTON, R. & REILLY, T. (1996). **Kinanthropometry and Exercise Physiology Laboratory Manual**. London, UK: E & FN SPON.
- BOUISSET, S. & ZATTARA, M. (1981). A Sequence of Postural Adjustments Precedes Voluntary Movements. **Neuroscience Letters**. 22: 263-270.
- BOUISSET, S. & ZATTARA, M. (1987). Biomechanical Study of the Programming of Anticipatory Postural Adjustments associated with Voluntary Movement. **Journal of Biomechanics**. 20: 735-742.
- BRINCKMANN, P., BIGGEMANN, M. & HILWEG, D. (1989). Prediction of the Compressive Strength of Human Lumbar Vertebrae. **Clinical Biomechanics**. 4 (suppl. 2).
- BRODKE, D.S. & RITTER, S.M. (2005). Nonsurgical Management of Low Back Pain and Lumbar Disk Degeneration. **AAOS Instructional Course Lectures**. 54: 279-286.
- BROOKE, M.H. & KAISER, K.K. (1970). Muscle Fibre Types: How Many and What Kind? **Archives of Neurology**. 23: 369-379.
- BROX, J.I., SORENSEN, R., FRIIS, A., NYGAARD, O., INDAHL, A., KELLER, A., INGEBRIGTSEN, T., ERIKSEN, H.R., HOLM, I., KOLLER, A.K., RIISE, R. & REIKERÅS, O. (2003). Randomized Clinical Trial of Lumbar Instrumented Fusion and Cognitive Intervention and Exercise in Patients with Chronic Low Back Pain and Disc Degeneration. **Spine**. 28(17): 1913-1921.
- BRUMAGNE, S., CORDO, P., LYSSENS, R., VERSCHUEREN, S. & SWINNEN, S. (2000). The Role of Paraspinal Muscle Spindles in Lumbosacral Position Sense in Individuals with and without Low Back Pain. **Spine**. 25: 989-994.

- BUCHBINDER, R., JOLLEY, D. & WYATT, M. (2001). 2001 Volvo Award Winner in Clinical Studies: Effects of a Media Campaign on Back Pain Beliefs and its Potential Influence on Management of Low Back Pain in General Practice. ***Spine***. 26: 2535-2542.
- BURGESS, P.R., WEI, J.Y., CLARK, F.J. & SIMON, J.(1992). Signaling of Kinesthetic Information by Peripheral Sensory Receptors. ***Annual Review of Neuroscience***. 5: 171-187.
- BURNS, J.W., MULLEN, J.T., HIGDON, L.J., WEI, J.M. & LANSKY, D. (2000). Validity of the Pain Anxiety Symptoms Scale (PASS): Prediction of Physical Capacity Variables. ***Pain***. 84: 247-252.
- BURTON, A.K., ERIKSEN, H.R., LECLERC, A., BALAUÉ, F., HENROTIN, Y., MÜLLER, G., CARDON, G., LAHAD, A. & VAN DER BEEK, A.J. (2004). European Guidelines for Prevention in Low Back Pain. ***COST B13 Working Group on Guidelines for Prevention in Low Back Pain***.
- BURTON, A.K. (2005). How to Prevent Low Back Pain: On Behalf of the COST B13 Working Group on European Guidelines for Prevention in Low Back Pain. ***Best Practice & Research: Clinical Rheumatology***. 19(4): 541-555.
- CALLAGHAN, J.P. & MCGILL, S.M. (1995). Muscle Activity and Low Back Loads under External Shear and Compressive Loading. ***Spine***. 20: 992-998.
- CALLAGHAN, J.P., GUNNING, J.L. & MCGILL, S.M. (1998). The Relationship between Lumbar Spine Load and Muscle Activity during Extensor Exercises. ***Physical Therapy***. 78: 8-18.



CARRAGEE, E.J., CHEN, Y., TANNER, C.M., HAYWARD, C., ROSSI, M. & HAGLE, C. (2000). Can Discography cause Long-Term Back Symptoms in Previously Asymptomatic Subjects? *Spine*. 25: 1803-1808.

CARAGGEE, E.J. (2005). Persistent Low Back Pain. *New England Journal of Medicine*. 352:1891-1898.

CARR, D.B. & GOUDAS, L.C. (1999). Acute Pain. *The Lancet*. 353: 2051-2058.

CARREON, L.Y., GLASSMAN, S.D. & HOWARD, J. (2008). Fusion and Non-Surgical Treatment for Symptomatic Lumbar Degenerative Disease: A Systematic Review of Oswestry Disability Index and MOS Short Form-36 Outcomes. *The Spine Journal*. 8: 747-755.

CAREY, T.S., GARRETT, J.M., JACKMAN, A. & HADLER, N. (1999). Recurrence and Care Seeking after Acute Back Pain: Results of a Long-Term Follow-Up Study. *Medical Care*. 37: 157-164.

CAREY, T.S., GARRETT, J.M. & JACKMAN, A.M. (2000). Beyond the Good Prognosis. Examination of an Inception Cohort of Patients with Chronic Low Back Pain. *Spine*. 21: 115-120.

CAHMAK, A., YUCEL, B., OZYALCH, S.N., BAYRAKTAR, B., URAL, H.I., DURUOZ, M.T. & GENÇ, A. (2004). The Frequency and Associated Factors of LBP among a Younger Population in Turkey. *Spine*. 29: 1567-1572.

CHAPMAN, C.R. & GAVRIN, J. (1999). Suffering: The Contributions of Persistent Pain. *Lancet*. 353: 2233-2237.

- CHELBERG, M.K., BANKS, G.M., GEIGER, D.F. & OEGEMA, T.R. (1995). Identification of Heterogenous Cell Populations in Normal Human Intervertebral Disc. ***Journal of Anatomy***. 186: 43-53.
- CHERKIN, D.C., DEYO, R.A., STREET, J.H. & BARLOW, W. (1996). Predicting Poor Outcomes for Back Pain seen in Primary Care using Patients' Own Criteria. ***Spine***. 21: 2900-2907.
- CHILDS, J., FRITZ, J., FLYNN, T., IRGANG, J., JOHNSON, K., MAJKOWSKI, G. & DELITTO, A. (2004). A Clinical Predication Rule to Identify Patients with Low Back Pain Most Likely to Benefit from Spinal Manipulation: A Validation Study. ***Annals of Internal Medicine***. 141(12): 920-928, W165-W166.
- CHOLEWICKI, J. & MCGILL, S.M. (1992). Lumbar Posterior Ligament Involvement during Extremely Heavy Lifts Estimated from Fluoroscopic Measurements. ***Journal of Biomechanics***. 25: 17-28.
- CHOLEWICKI, J. & MCGILL, S.M. (1996). Mechanical Stability of the In Vivo Lumbar Spine: Implications for Injury and Chronic Low Back Pain. ***Clinical Biomechanics***. 11: 1-15.
- CHOLEWICKI, J., PANJABI, M.M. & KHACHATRYAN, A. (1997). Stabilizing Function of Trunk Flexor-Extensor Muscles around a Neutral Spine Posture. ***Spine***. 22: 2207-2212.
- CLELAND, J.A., CHILDS, J.D., PALMER, J.A. & EBERHART, S. (2005). Slump Stretching in the Management of Non-Radicular Low Back Pain: A Pilot Clinical Trail. ***Manual Therapy***.

- CLINGHAM, J.M. & HILLIARD, D.V. (1987). Some Personality Characteristics of the Super-Adherer: Following those Who Go Beyond Fitness. ***Journal of Sports Behaviour***. 10: 123-136.
- COHEN, J.E., GOEL, V., FRANK, J.W., BOMBARDIER, C., PELOSO, P. & GUILLEMIN, F. (1994). Group Education Interventions for People with Low Back Pain: An Overview of the Literature. ***Spine***. 19: 1214-1222.
- COLLINS, J.J. & DELUCA, C.J. (1993). Open-Loop and Closed-Loop Control of Posture: A Random-Walk Analysis of Center of Pressure. ***Experimental Brain Research***. 95: 308-318.
- COPPE, M.H., MARANI, E., THOMEER, R.T., OUDEGA, M. & GROEN, G.J. (1990). Innervation of Annulus Fibrosis in Low Back Pain. ***Lancet***. 336: 189-190.
- CORDO, P.J. & NASHNER, L.M. (1982). Properties of Postural Adjustments Associated with Rapid Arm Movements. ***Journal of Neurophysiology***. 47: 287-302.
- CRAM, R. & STEGER, J. (1983). EMG Scanning in Diagnosis of Chronic Pain. ***Biofeedback Self Regular***. 8: 229-241.
- CRESSWELL, A.G., GRUNDSTROM, H. & THORSTENSSON, A. (1992). Observations on Intra-Abdominal Pressure and Patterns of Abdominal Intra-Muscular Activity in Man. ***Acta Physiologica Scandinavica***. 144: 409-418.
- CRESSWELL, A.G., ODDSSON, L. & THORSTENSSON, A. (1994). The Influence of Sudden Perturbation on Trunk Muscle Activity and Intra-Abdominal Pressure while Standing. ***Experimental Brain Research***. 98: 336-341.

- CRESSWELL, A. & THORSTENSSON, A. (1994). Changes in Intra-Abdominal Pressure, Trunk Muscle Activation and Force during Isokinetic Lifting and Lowering. ***European Journal of Applied Physiology***. 68: 315-321.
- CRISCO, J.J. & PANJABI, M.M. (1991). The Intersegmental and Multisegmental Muscles of the Lumbar Spine: A Biomechanical Model Comparing Lateral Stabilization Potential. ***Spine***. 16: 793-799.
- CRISCO, J.J., PANJABI, M.M., YAMAMOTO, I. & OXLAND, T.R. (1992). Euler Stability of the Human Ligamentous Lumbar Spine. Part II: Experiment. ***Clinical Biomechanics***. 7: 27-32.
- CROFT, P., PAPAGEORGIOU, A. & MCNALLY, R. (1997). Low Back Pain. In STEVENS, A. & RAFTERY, J. eds. **Health Care Needs Assessment. Second Series: The Epidemiologically Based Needs Assessment Reviews**. Oxford, NY: Radcliffe Medical Press.
- CROFT, P.R., MACFARLANE, G.J., PAPAGEORGIOU, A.C., THOMAS, E. & SILMAN, A.J. (1998). Outcome of Low Back Pain in General Practice: A Prospective Study. ***BMJ***. 316: 1356-1359.
- CROFT, P.R., PAPAGEORGIOU, A.C., THOMAS, E., MACFARLANE, G.J. & SILMAN, A.J. (1999). Short-Term Physical Risk Factors for New Episodes of LBP. Prospective Evidence from the South Manchester Back Pain Study. ***Spine***. 24: 1556-1561.
- CROMBEZ, G., VLAEYEN, J.W., HEUTS, P.H. & LYSENS, R. (1999). Pain-Related Fear is More Disabling than Pain Itself: Evidence on the Role of Pain-Related Fear in Chronic Back Pain Disability. ***Pain***. 80: 329-339.

- CUMMINS, T.R., BLACK, J.A., DIB-HAJJ, S.D.& WAXMAN, S.G. (2000). Glial-derived Neurotrophic Factor Upregulates Expression of Functional SNS and NaN Sodium Channels and their Currents in Axotomized Dorsal Root Ganglion Neurons. ***Journal of Neuroscience***. 20: 8754-8761.
- CUNHA, F.Q. & FERREIRA, S.H. (2003). Peripheral Hyperalgesic Cytokines. ***Advances in Experimental Medicine and Biology***. 521: 22-39.
- DANNEELS, L.A., VANDERSTRAETEN, G.G.&CAMBIER, D.C., WITVROUW, E.E., BOURQOIS, J., DANKAERTS, W. & DE CUYPER, H.J. (2001). Effects of Three Different Modalities on the Cross-Sectional Area of the Lumbar Multifidus Muscle in Patients with Chronic Low Back Pain. ***British Journal of Sports Medicine***. 35: 186-191.
- DAWES, H.N., BARKER, K.L., COCKBURN, J., ROACH, N., SCOTT, O. & WADE, D. (2005). Borg's Rating of Perceived exertion Scales: Do the Verbal Anchors Mean the Same for Different Clinical Groups? ***Archives of Physical Medicine and Rehabilitation***. 86: 912-916.
- DELAVIER, F. (2001). ***Strength Training Anatomy***. Champaign, IL: Human Kinetics.
- DEMOULIN, C., VANDERTHOMMEN, M., DUYSSENS, C. & CRIELAARD, J.M. (2004). Spinal Muscle Evaluation Using the Sorensen Test: A Critical Appraisal of the Literature. ***Joint Bone Spine***. 73(2006): 43-50.
- DESMEULES, J.A., CEDRASCHI, C., RAPITI, E., BAUMGARTNER, E., FINKH, A., COHEN, P., DAYER, P. & VISCHER, T.L.(2003). Neurophysiologic Evidence for a Central Sensitization in Patients with Fibromyalgia. ***Arthritis and Rheumatism***. 48: 1420-1429.

- DE VET, H.C.W., HEYMANS, M.W., DUNN, K.M., POPE, D.P., VAN DER BEEK, A.J., MACFARLANE, G.J, BOUTER, L.M. & CROFT, P.R. (2002). Episodes of Low Back Pain: A Proposal for Uniform Definitions to be used in Research. *Spine*. 27: 2409-2416.
- DEYO, R.A. (1982). Compliance with Therapeutic Regimens in Arthritis: Issues, Current Status, and a Future Agenda. *Seminars in Arthritis and Rheumatism*. 12: 233-244.
- DEYO, R.A., WALSH, N.E., MARTIN, D.C., SCHOENFELD, L.S. & RAMAMURTHY, S. (1990). A Controlled Trial of Transcutaneous Electrical Nerve Stimulation (TENS) and Exercise for Chronic Low Back Pain. *The New England Journal of Medicine*. 322(23): 1627-1634.
- DEYO, R.A. (1994). Magnetic Resonance Imaging. Terrific Test or Tar Baby? *New England Journal of Medicine*. 331: 115-116.
- DEYO, R.A. (1996). Acute Low Back Pain: A New Paradigm for Management: Limited Imaging and Early Return to Normal Activities. *BMJ*. 313; 1343-1345.
- DEYO, R.A. & PHILLIPS, W.R. (1996). Low Back Pain: A Primary Health Challenge. *Spine*. 21: 2826-2832.
- DEYO, R.A., BATTIE, M., BEURSKENS, A., BOMBARDIER, C., CROFT, P., KOES, B., MALMIVAARA, A., ROLAND, M., VAN KORFF, M. & WADDELL, G. (1998). Outcome Measures for Low Back Pain Research: A Proposal for Standardized Use. *Spine*. 23: 2003-2013.

- DEYO, R.A, MIRZA, S. & MARTIN, B. (2006). Back Pain Prevalence and Visit Rates: Estimates from US National Surveys, 2002. *Spine*. 31: 2724-2727.
- DIATCHENKO, L, NACKLEY, A.G., SLADE, G.D., FILLINGIN, R.B. & MAIXNER, W. (2006). Idiopathic Pain Disorders – Pathways of Vulnerability. *Pain*. 123: 226-230.
- DINGWELL, J.B. & CUSUMANO, J.P. (2000). Nonlinear Time Series Analysis of Normal and Pathological Human Walking. *Chaos*. 10: 848-863.
- DIONNE, C., KOEPESELL, T.D., VON KORFF, M., DEYO, R.A., BARLOW, W.I. & CHECKOWAY, H. (1995). Formal Education and Back-Related Disability: In Search of an Explanation. *Spine*. 20: 2721-2750.
- DIONNE, C., KOEPESELL, T.D., VON KORFF, M., DEYO, R.A., BARLOW, W.E. & CHECKOWAY, H. (1997). Predicting Long-Term Functional Limitations among Back Pain Patients in Primary Care Settings. *Journal of Clinical Epidemiology*. 50: 31-43.
- DISHMAN, R.K. (1991). Increasing and Maintaining Exercise and Physical Activity. *Behavior Therapy*. 22: 345-378.
- DOFFERHOF, A.S.M. & VINK, P. (1985). The Stabilising Function of the mm. Iliocostales and the mm. Multifidi during Walking. *Journal of Anatomy*. 140: 329-336.
- DOLAN, P. & ADAMS, J. (1993). The Relationship between EMG Activity and Extensor Moment Generation in the Erector Spinae Muscles during Bending and Lifting Activities. *Journal of Biomechanics*. 26: 13-22.

- DOLEYS, D.M., KLAPOW, J.C. & HAMMER, M. (1997). Psychological Evaluation in Spinal Cord Stimulation Therapy. *Pain Review*. 4: 189-207.
- DONCHIN, M., WOOLF, O., KAPLAN, L. & FLOMAN, Y. (1990). Secondary Prevention of Low Back Pain. A Clinical Trial. *Spine*. 15: 1317-1320.
- DONISCH, E.W. & BASMAJIAN, J.V. (1972). Electromyography of Deep Back Muscles in Man. *The American Journal of Anatomy*. 133: 15-36.
- D'ORAZIO, B.P. (Editor). (1999). **Low Back Pain Handbook**. Boston, MA: Butterworth-Heinemann.
- DUPUIS, P.R., YONG-HING, K., CASSIDY, J.D. & KIRKALDITY-WILLIS. (1985). Radiographic Diagnosis of Degenerative Lumbar Spinal Instability. *Spine*. 10: 262-276.
- DVORAK, J., PANJABI, M.M., CHANG, D.G., THEILER, R. & GROB, D. (1991). Functional Radiographic Diagnosis of Degenerative Lumbar Spine. Flexion-Extension and Lateral Bending. *Spine*. 16: 562-571.
- DWORKIN, R.H. & BREITBART, W.S. eds. (2004). **Psychosocial Aspects of Pain: A Handbook for Healthcare Professionals**. Seattle, WA: IASP Press.
- EDGAR, M.A. & PARK, W.M. (1974). Induced Patterns on Passive Straight-Leg Raising in Lower Limb Disc Protrusion. *Journal of Bone and Joint Surgery British Volume*. 56: 658-666.



- EDGERTON, V., WOLF, S., LEVENDOWSKI, D. & ROY, R. (1996). Theoretical Basis for Patterning EMG Amplitudes to Assess Muscle Dysfunction. ***Medicine and Science in Sports and Exercise***. 28: 744-751.
- EISENBERGER, N.I., LIEBERMAN, M.D. & WILLIAMS, K.D. (2003). Does Rejection Hurt? An fMRI Study of Social Exclusion. ***Science***. 302: 290-292.
- ELDERS, L.A., VAN DER BEEK, A.J. & BURDORF, A. (2000). Return to Work after Sickness Absence due to Back Disorders – A Systematic Review on Intervention Strategies. ***International Archives of Occupational and Environmental Health***. 73: 339-348.
- ELFERING, A., SEMMERM, N., BIRKHOFFER, D., ZANETTI, M., HODLER, J. & BOOS, N. (2002). Risk Factors for Lumbar Disc Degeneration. A 5-Year Prospective MRI Study in Asymptomatic Individuals. ***Spine***. 27: 125-134.
- ENOKA, R.M. (1994). ***Neuromechanical Basis of Kinesiology***. (2<sup>nd</sup> Edition). Champaign, IL: Human Kinetics.
- EPPING, J.J.E., WAHLGREN, D.R., WILLIAMS, R.A., PRUITT, S.D., SLATER, M.A., PATTERSON, T., GRANT, I., WEBSTER, J.S. & ATKINSON, J.H. (1998). Transition to Chronic Pain in Men with Low Back Pain: Predictive Relationship among Pain Intensity, Disability and Depressive Symptoms. ***Health Psychology: Official Journal of The Division of Health Psychology, American Psychological Association***. 17: 421-427.
- EPSTEIN, J.A., EPSTEIN, B.S., ROSENTHAL, A.D., CARRAS, R. & LAVINE, L.S. (1972). Sciatica caused by Nerve Root Entrapment in the Lateral Recess: The Superior Facet Syndrome. ***Journal of Neurosurgery***. 36: 584-589.

- ERHARD, R.E., DELITTO, A. & CIBULKA, M.T. (1994). Relative Effectiveness of an Extension Program and Combined Program of Manipulation and Flexion and Extension Exercises in Patients with Acute Low Back Syndrome. *Physical Therapy*. 74: 1093-1100.
- ERRINGTON, R.J., PUUSTJARVI, K., WHITE, I.R., ROBERTS, S. & URBAN, J.P. (1998). Characterisation of Cytoplasm-Filled Processes in Cells of the Intervertebral Disc. *Journal of Anatomy*. 192: 369-378.
- EVERILL, B. & KOCSIS, J.D. (1999). Reduction in Potassium Currents in Identified Cutaneous Afferent Dorsal Root Ganglion Neurons after Axotomy. *Journal of Neurophysiology*. 82: 700-708.
- EYRE, D.R. & MUIR, H. (1977). Quantitative Analysis of Types I and II Collagens in the Human Intervertebral Disc at Various Ages. *Biocimica Biophysica Acta*. 492: 29-42.
- FAAS, A., CHAVANNES, A.W. & GUBBELS, J.W. (1993). A Randomized, Placebo-Controlled Trail of Exercise Therapy in Patients with Acute Low Back Pain. *Spine*. 18: 1388-1395.
- FAAS, A., VAN EIJK, J.T.H.M., CHAVANNES, A.W. & GUBBELS, J.E. (1995). A Randomized Trail of Exercise Therapy in Patients with Acute Low Back Pain. *Spine*. 20(8): 941-947.
- FAIRBANK, J.C.T. & DAVIES, J.B. (1980). The Oswestry Low Back Pain Disability Questionnaire. *Physiotherapy*. 66: 271-273.

FAIRBANK, J.C.T & PYNSENT, P.B. (2000). The Oswestry Disability Index. **Spine**. 25(22): 2940-2953.

FALCONER, M.A., MCGEORGE, M. & BEGG, A.C. (1948). Observation on the Cause and Mechanism of Symptom-Reproduction in Sciatica and Low Back Pain. **Journal of Neurology, Neurosurgery and Psychiatry**. 11: 13-26.

FARFAN, H.F. (1973). **Mechanical Disorders of the Low Back**. Philadelphia, PA: Lea and Febiger.

FATHALLAH, F.A., MARRAS, W.S. & PARNAINPOUR, M. (1998). The Role of Complex, Simultaneous Trunk Motions in the Risk of Occupation-Related Low Back Disorders. **Spine**. 23: 1035-1042.

FERGUSON, S.A., MARRAS, W.S. & GUPTA, P. (2000). Longitudinal Quantitative Measures of the Natural Course of Low Back Pain Recovery. **Spine**. 25: 1950-1956.

FEISE, R.J. & MENKE, J.M. (2001). Functional Rating Index: A New Valid and Reliable Instrument to Measure the Magnitude of Clinical Change in Spinal Conditions. **Spine**. 26(1): 78-87.

FIDLER, M.W., JOWETT, R.L. & TROUP, J.D. (1975). Myosin ATPase Activity in Multifidus Muscle from Cases of Lumbar Spine Derangement. **The Journal of Bone and Joint Surgery. British Volume**. 57: 220-227.

FISHER, K. & JOHNSTON, M. (1997). Validation of the Oswestry Low Back Pain Disability Questionnaire, its Sensitivity as a Measure of Change following Treatment and its

Relationship with other Aspects of the Chronic Pain Experience. ***Physiotherapy Theory and Practice***. 13: 67-80.

FITTS, P.M. (1954). The Information Capacity of the Human Motor System in Controlling the Amplitude of Movement. ***Journal of Experimental Psychology***. 47: 381-391.

FLOR, H. (2003). Cortical Reorganization and Chronic Pain: Implications for Rehabilitation. ***Journal of Rehabilitation Medicine***. 66-72.

FLOR, H., NIKOLAJSEN, L. STAEHELIN-JENSEN, T. (2006). Phantom Limb Pain: A Case of Maladaptive CNS Plasticity? ***Nature Reviews. Neuroscience***. 7: 873-881.

FLOYD, W.F. & SILVER, P.H.S. (1951). Function of the Erector Spinae in Flexion of the Trunk. ***Lancet***. 133-134.

FORBES, J.L. (1986). Training Lessons Learned from Peak Performance Episodes. ***US Army Research Institute for the Behavioral and Social Sciences Report***. Jun Tech Rpt 711: 29.

FORDYCE, W.E. (1995). ***Back Pain in the Workplace: Management of Disability in Nonspecific Conditions***. Seattle, WA: IASP Press.

FOREMAN, J.C. (1987). Peptides and Neurogenic Inflammation. ***British Medical Bulletin***. 43: 386-400.

FORWELL, L.A. & CARNAHAN, H. (1996). Proprioception during Manual Aiming in Individuals with Shoulder Instability and Controls. ***The Journal of Orthopaedic and Sports Physical Therapy***. 23: 111-119.

- FRANK, J., KERR, M.S., BROOKER, A-S., DEMAIO, S.E., MAETZEL, A., SHANNON, H.S., SULLIVAN, T.J., NORMAN, R.W. & WELLS, R.P. (1996). Disability Resulting from Occupational Low Back Pain. Part I: What do we know about Primary Prevention? A Review of the Scientific Evidence on Prevention before Disability Begins. **Spine**. 21: 2908-2917.
- FRANK, J., SINCLAIR, S., HOGG-JOHNSON, S., SHANNON, H., BOMBARDIER, C., BEATON, D. & COLE, D. (1998). Preventing Disability from Work-Related Low Back Pain. New Evidence Gives New Hope – If We Can Just Get All the Players Onside. **CMAJ: Canadian Medical Association Journal**. 158: 1625-1631.
- FREEMONT, A.J., PEACOCK, T.E., GOUPILLE, P., HOYLAND, J.A., O'BRIEN, J. & JAYSON, M.I.V. (1997). Nerve In-Growth into Diseased Intervertebral Disc in Chronic Back Pain. **The Lancet**. 350: 178-181.
- FRIBERG, O. (1987). Lumbar Instability: A Dynamic Approach by Traction-Compression Radiography. **Spine**. 12: 119-129.
- FRIEDLI, W.G., COHEN, L., HALLET, M., STANHOPE, S. & SIMON, S.R. (1988). Postural Adjustments Associated with Rapid Voluntary Arm Movements: II. Biomechanical Analysis. **Journal of Neurology, Neurosurgery, and Psychiatry**. 51: 232-243.
- FRIEDRICH, M., GITTLER, G., ARENDASY, M. & FRIEDRICH, K.M. (2005). Long-Term Effect of a Combined Exercise and Motivational Program on the Level of Disability of Patients with Chronic Low Back Pain. **Spine**. 30(9): 995-1000.
- FRITZ, J.M., ERHARD, E.R. & HAGEN, B.F. (1998). Segmental Instability of the Lumbar Spine. **Physical Therapy**. 78: 889-896.

- FRITZ, J.M., GEORGE, S.Z. & DELITTO, A. (2001). The Role of Fear-Avoidance Beliefs in Acute Low Back Pain: Relationships with Current and Future Disability and Work Status. *Pain*. 94: 7-15.
- FRITZ, J.M. & GEORGE, S.Z. (2002). Identifying Psychosocial Variables in Patients with Acute Work-Related Low Back Pain: The Importance of Fear-Avoidance Beliefs. *Physical Therapy*. 82: 973-983.
- FRONTERA, W.R., MEREDITH, C.N., O'REILLY, K.P. (1988). Strength Conditioning in Older Men: Skeletal Muscle Hypertrophy and Improved Function. *Journal of Applied Physiology*. 64: 1038-1044.
- FRYMOYER, J.W., POPE, M.H., CLEMENTS, J.H., WILDER, D.G., MACPHERSON, B. & ASHIKAGA, T. (1983). Risk Factors in Low Back Pain. An Epidemiological Survey. *The Journal of Bone and Joint Surgery*. 65A: 213-218.
- FRYMOYER, J.W. & SELBY, D.K. (1985). Segmental Instability: Rationale for Treatment. *Spine*. 10: 280-286.
- FRYMOYER, J.W. & CATS-BARIL, W.L. (1991). An Overview of the Incidence and Costs of Low Back Pain. *The Orthopedic Clinics of North America*. 2: 263-271.
- FRYMOYER, J.W. & CATS-BARIL, W.L. (1991). An Overview of the Incidences and Costs of Low Back Pain. *The Orthopedic Clinics of North America*. 22: 263-271.
- FRYMOYER, J.W. (1992). Predicting Disability from Low Back Pain. *Clinical Orthopedics*. 101-109.

FURLAN, A.D., CLARKE, J., ESMAIL, R., SINCLAIR, S., IRVIN, E. & BOMBARDIER, L. (2001). A Critical Review of Reviews on the Treatment of Chronic Low Back Pain. *Spine*. 26: E155-162.

FYHRIE, D.P. & SCHAFFLER, M.B. (1994). Failure Mechanisms in Human Vertebral Cancellous Bone. *Bone*. 15(1): 105-109.

GATCHEL, R.J., POLATIN, P.B. & KINNEY, R.K. (1995). Predicting Outcome of Chronic Back Pain Using Clinical Predictors of Psychopathology: A Prospective Analysis. *Health Psychology*. 14: 415-420.

GALANTINO, M.L., BZDEWKA, T.M., EISSLER-RUSSON, J.L., HOLBROOK, M.L., MOGCK, E.P., GEIGLE, P. & FARRAR, J.T. (2004). The Impact of Modified Hatha Yoga on Chronic Low Back Pain: A Pilot Study. *Alternative Therapies in Health and Medicine*. 10: 56-59.

GALLOIS, J. & JAPOIT, T. (1925). Architecture Intérieure des Vertèbres du Point de vue Statique et Physiologique. *Revue de Chirurgi (Paris)*. 63 : 688.

GANDEVIA, S.C., MCCLOSKEY, D.L. & BURKE, D. (1992). Kinesthetic Signals and Muscle Contraction. *Trends In Neurosciences*. 15: 62-65.

GARDNER-MORSE, M., STOKES, IAF. & LAIBLE, J.P. (1995). Role of Muscles in Lumbar Spine Stability in Maximum Extension Efforts. *Journal of Orthopaedic Research*. 13: 802-808.

GARDNER-MORSE, M.G. & STOKES, I.A. (1998). The Effects of Abdominal Muscle Coactivation on Lumbar Spine Stability. *Spine*. 23: 86-92.

- GEISSER, M.E., ROTH, R.S., THEISEN, M.E., ROBINSON, M.E. & RILEY, J.L. (2000). Negative Affect, Self-Report of Depressive Symptoms and Clinical Depression: Relation to the Experience of Chronic Pain. ***Clinical Journal of Pain***. 16: 110-120.
- GEISSER, M.E., WIGGERT, E.A., HAIG, A.J. & COLWELL, M.O. (2005). A Randomized, Controlled Trial of Manual Therapy and Specific Adjuvant Exercises for Chronic Low Back Pain. ***Clinical Journal of Pain***. 21: 463-470.
- GILGIL, E., KACAR, C., BÜTÜN, B., TUNCER, T., URHAN, S., YILDIRIM, C., SÜNBÜLOGLU, G., ARIKAN, V., TEKEOGLU, I., ÖKSÜZ, M. & DÜNDAR, U. (2005). Prevalence of Low Back Pain in a Developing Urban Setting. ***Spine***. 30: 1093-1098.
- GODDARD, M.D. & REID, J.D. (1965). Movements Induced by Straight Leg Raising in the Lumbo-Sacral Roots, Nerves and Plexus, and in the Intrapelvic Section of the Sciatic Nerve. ***Journal of Neurology, Neurosurgery, and Psychiatry***. 28: 12.
- GOEKEN, L.N. & HOF, A.L. (1991). Instrumental Straight Leg Raising: A New Approach to Lasegue's Test. ***Archives of Physical Medicine and Rehabilitation***. 72: 959-966.
- GOEKEN, L.N. & HOF, A.L. (1993). Instrumental Straight Leg Raising: Results in Healthy Subjects. ***Archives of Physical Medicine and Rehabilitation***. 74: 194-203.
- GOEKEN L.N. & HOF, A.L. (1994). Instrumental Straight Leg Raising: Results in Patients. ***Archives of Physical Medicine and Rehabilitation***. 75: 470-477.



- GOEL, V.K., KONG, W., HAN, J.S., WEINSTEIN, J.N. & GILLBERTSON, L.G. (1993). A Combined Finite Element and Optimization of Lumbar Spine Mechanics with and without Muscles. *Spine*. 18: 1531-1541.
- GOLDBY, L.J., MOORE, A.P., DOUST, J.&TREW, M.E. (2006). A Randomized Control Trial Investigating the Efficiency of Musculoskeletal Physiotherapy on Chronic Low Back Pain Disorders. *Spine*. 31(10): 1083-1093.
- GRABINER, M., KOH, T. & GHAZAWI, A.E. (1992). Decoupling of Bilateral Paraspinal Excitation in Subjects with Low Back Pain. *Spine*. 17: 1219-1223.
- GRACOVETSKY, S., FARFAN, H. & HELLEUR, C. (1985). The Abdominal Mechanism. *Spine*. 10: 317-324.
- GRANATA, K.P. & MARRAS, W.S. (1993). An EMG-Assisted Model of Loads on the Lumbar Spine during Asymmetric Trunk Extension. *Journal of Biomechanics*. 26: 1429-1438.
- GRANATA, K.P. & MARRAS, W.S. (1995). The Influence of Trunk Muscle Coactivity upon Dynamic Spinal Loads. *Spine*. 20: 913-919.
- GRANATA, K.P. & ORISHIMO, K. (2001). Response of Trunk Muscle Coactivation to Changes in Spinal Stability. *Journal of Biomechanics*. 34: 1117-1123.
- GRANATA, K.P. & WILSON, S.E. (2001). Trunk Posture and Spinal Stability. *Journal of Biomechanics*. 16: 650-659.

- GRANATA, K.P.&ENGLAND, S.A. (2006). Stability of Dynamic Trunk Movement. **Spine**. 31(10): E271—E276.
- GRAVES, J.E., POLLOCK, M.L., FOSTER, D., LEGGETT, S.H., CARPENTER, D.M., VUOSO, R. & JONES, A. (1990). Effect of Training Frequency and Specificity on Isometric Lumbar Extension Strength. **Spine**. 15: 504-509.
- GREENNOUGH, C.G., OLIVER, C.W. & JONES, A.P.C. (1998). Assessment of Spinal Musculature using Surface Electromyographic Spectral Colour Mapping. **Spine**. 23: 1768-1774.
- GRILLNER, S., NILSSON, J. & THORSTENSSON, A. (1978). Intraabdominal Pressure Changes during Natural Movements in Man. **Acta Physiologica Scandinavica**. 103: 275-283.
- GRÖNBLAD, M., WEISTEIN, J.N. & SANTAVIERA, S. (1991). Immunohistochemical Observations on Spinal Tissue Innervation: A Review of Hypothetical Mechanisms of Back Pain. **Acta Orthopaedica Scandinavica**. 62: 614-622.
- GRØNNINGSÆTER, H., HYTTEN, K., SKAULI, G., CHRISTENSEN, C.C. & URSIN, H. (1992). Improved Health and Coping by Physical Exercise or Cognitive Behavioral Stress Management Training in a Work Environment. **Psychology & Health**. 7: 147-163.
- GROSS, M.T. (1987). Effects of Recurrent Lateral Ankle Sprains on Active and Passive Judgments of Joint Position. **Physical Therapy**. 67: 1505-1509.
- GROTLE, M., VØLLESTAD, N.K., VEIERØD, M. & BROX, J.I. (2004). Fear-Avoidance Beliefs and Distress in Relation to Disability in Acute and Chronic Low Back Pain: A

Cross-Sectional Comparison of Relationships with Activity Limitations and Work Restrictions. *Pain*. 112(3): 343-352.

GROTLE, M., BROX, J.I., GLOMSRØD, B., VEIERØD, M.B., LØNN, J.H. & VØLLESTOD, N.K. (2005). Clinical Course and Prognostic Factors in Acute Low Back Pain: Patients Consulting Primary Care for the First Time. *Spine*. 30: 976-982.

GROTLE, M., VØLLESTAD, N.K. & BROX, J.I. (2006). Clinical Course and Impact of Fear-Avoidance Beliefs in Low Back Pain: Prospective Cohort Study of Acute and Chronic Low Back Pain: II. *Spine*. 31(9): 1038-1046.

GRUBB, B.D., STILLER, R.U. & SCHAIBLE, H.G. (1993). Dynamic Changes in the Receptive Field Properties of Spinal Cord Neurons with Ankle Input in Rats with Unilateral Adjuvant-Induced Inflammation in the Ankle Region. *Experimental Brain Research*. 92: 441-451.

GUNNING, J.L., CALLAGHAN, J.P. & MCGILL, S.M. (2001). The Role of Prior Loading History and Spinal Posture on the Compressive Tolerance and Type of Failure in the Spine using a Porcine Trauma Model. *Clinical Biomechanics*. 16(6): 471-480.

GRUSSER, S.M., MUHLNICKEL, W., SCHAEFER, M., VILLRINGER, K., CHRISTMANN, C., KOEPPE, C. & FLOR, H. (2004). Remote Activation of Referred Phantom Sensation and Cortical Reorganization in Human Upper Extremity Amputees. *Experimental Brain Research*. 154: 97-102.

GUREJO, O., VON KORFF, M., SIMON, G. & SATER, R. (1998). Persistent pain and Well-Being – A WHO Study in Primary Care. *JAMA*. 280: 147-151.

- GUZMAN, J., ESMAIL, R., KARJALAINEN, K., MALMIVAARA, A. IRVIN, E. & BOMBARDIER, C. (2001). Multidisciplinary Rehabilitation for Chronic Low Back Pain: Systematic Review. **BMJ**. 322: 1511-1516.
- HAGEN, E.M., ERIKSEN, H.R. & URSIN, H. (2000). Does Early Intervention with a Light Mobilization Program Reduce Long-Term Sick Leave for LBP. **Spine**. 25: 1973-1976.
- HAKKINEN, A., YLINEN, J., KAUTIAINEN, H., AIRAKSINEN, O., HERNO, A., TARVAINEN, U & KIVIRANTA, I. (2003). Pain, Trunk Muscle Strength, Spine Mobility and Disability following Lumbar Disc Surgery. **Journal of Rehabilitation Medicine**. 35: 236-240.
- HALL, H. & ICETON, J.A. (1983). BackSchool: An Overview with Specific Reference to the Canadian Back Education Units. **Clinical Orthopaedics**. 179: 10-17.
- HALL, H. MCINTOSH, G., MELLES, T., HOLOWAACHUK, B. & WAI, E. (1994). Effect of Discharge Recommendation on Outcome. **Spine**. 19: 2033-2037.
- HALL, T., ZUSMAN, M. & ELVEY, R. (1995). Manually Detected Impediments during the Straight Leg Raise Test. **MPAA Conference Proceedings 9<sup>th</sup> Biennial Conference**. 48-53.
- HALL, T., ZUSMAN, M. & ELVEY, R. (1998). Adverse Mechanical Tension in the Nervous System? Analysis of Straight Leg Raise. **Manual Therapy**. 3: 140-146.
- HAN, H.C., LEE, D.H. & CHUNG, J.M. (2000) Characteristics of Ectopic Discharges in Rat Neuropathic Pain Model. **Pain**. 84: 253-261.

- HANDWERKER, H.O. (1999). **Einführung in die Pathophysiologie des Schmerzes.** Berlin, Germany: Springer.
- HANSEN, J.W. (1964). Postoperative Management in Lumbar Disc Protrusions. I. Indications, Methods and Results. II. Follow-Up on a Trained and an Untrained Group of Patients. ***Acta Orthopaedica Scandinavica.*** 17 (supplement 71): 1-47.
- HARADA, M., ABUMI, K., ITO, M. & KANEDA, K. (2000). Cineradiographic Motion Analysis of Normal Lumbar Spine during Forward and Backward Flexion. ***Spine.*** 25: 1932-1937.
- HARREBY, M., HESSELSON, G., KJER, J. & NEERGAARD, K. (1997). LBP and Physical Exercise in Leisure Time in 38-Year-Old Men and Women: A 25-Year Prospective Cohort Study of 640 School Children. ***European Spine Journal: Official Publication of the European Spine Society, The European Spinal Deformity Society, and The European Section of The Cervical Spine Research Society.*** 6: 181-186.
- HARRIS, G.R. & SUSMAN, J.L. (2002). Managing Musculoskeletal Complaints with Rehabilitation Therapy: Summary of the Philadelphia Panel, Evidence Based Clinical Practice Guidelines on Musculoskeletal Rehabilitation Interventions. ***The Journal of Family Practice.*** 51: 1042-1046.
- HARRIS, R.E., CLAUW, D.J., SCOTT, D.J., MCLEAN, S.A., GRACEY, R.H., ZUBIETA, J.K. (2007). Decreased Central Muopioid Receptor Availability in Fibromyalgia. ***The Journal of Neuroscience: The Official Journal of the Society for Neuroscience.*** 27: 10000-10006.
- HARSTALL, C. & OSPINA, M. (2003). How Prevalent is Chronic Pain? ***Pain: Clinical Updates.*** 11: 1-4.

- HARTIGAN, C., RAINVILLE, J., SOBEL, J.B. & HIPONA, M. (2000). Long-Term Exercise Adherence after Intensive Rehabilitation for Chronic Low Back Pain. ***Medicine and Science in Sports and Exercise***. 32: 551-557.
- HASEGAWA, K., KITAHARA, K., HARA, T., TAKANO, K., SHIMODA, H. & HOMMA, T. (2008). Evaluation of Lumbar Segmental Instability in Degenerative Diseases by Using a New Intraoperative Measurement System. ***Journal of Neurosurgery: Spine***. 8: 255-262.
- HAWLEY, J.A. (2009). Molecular Responses to Strength and Endurance Training: Are They Incompatible. ***Applied Physiology, Nutrition and Metabolism***. 34(3): 355-361.
- HAYDEN, J.A., VAN TULDER, M.W., MALMIVAARA, A.V. & KOES, B.W. (2005). Meta-Analysis: Exercise Therapy for Non-Specific Low Back Pain. ***Annals of Internal Medicine***. 142: 765-775.
- HELLANDSIG, E.T. (1998). Motivational Predictors of High Performance and Discontinuation in Different Types of Sport among Talented Teenage Athletes. ***International Journal of Sports Psychology***. 29: 27-44.
- HERBISON, G.J., JAWEED, M.M. & DITUNNO, J.F. (1982). Muscle Fiber Types. ***Archives of Physical Medicine and Rehabilitation***. 63: 227-230.
- HERRINGTON, L., BENDIX, K., CORNWELL, C., FIELDEN, N. & HANKEY, K. (2007). What is the Normal Response to Structural Differentiation within the Slump and Straight Leg Raise Tests? ***Manual Therapy***. doi:10.1016/j.math.2007.01.013.

- HESTBAEK, L., LEBOEUF-YDE, C., ENGBERG, M., LAURITZEN, T., BRUUN, N.H.& MANNICHE, C. (2003a). The Course of Low Back Pain in a General Population. Results from a 5-Year Prospective Study. ***Journal of Manipulative and Physiological Therapeutics***. 26: 213-219.
- HESTBAEK, L., LEBOEUF-YDE, C. & MANNICHE, C. (2003b). Low Back Pain: What is the Long-Term Course? A Review of Studies of General Patient Populations. ***European Spine Journal***. 12: 149-165.
- HEYMANS, M.W., VAN TULDER, M.W., ESMAIL, R., BOMBARDIER, C. & KOES, B.W. (2004). Back Schools for Non-Specific Low Back Pain (Cochrane Review) ***Cochrane Database System Review*** (4): CD000261.
- HEYMANS, M.W., VAN TULDER, M.W., ESMAIL, R., BOMBARDIER, C. & KOES, B.W. (2005). Back Schools for Nonspecific Low Back Pain: A Systematic Review within the Framework of the Cochrane Collaboration Back Review Group. ***Spine***. 30: 2153-2163.
- HEYMANS, M.W., DE VET, H.C.W., BONGERS, P.M., KNOL, D.L., KOES, B.W. & MECHELEN, W. (2006). The Effectiveness of High-Intensity versus Low-Intensity Back Schools in an Occupational Setting. ***Spine***. 31(10): 1075-1082.
- HIDES, J.A, JULL, G.A.&RICHARDSON, C.A. (2001). Long-Term Effects of Specific Stabilizing Exercises for First Episode Low Back Pain. ***Spine***. 26: E243-248.
- HIDES, J.A., STOKES, M.J., SAIDE, M., JULL, G. & COOPER, D. (1994). Evidence of Lumbar Multifidus Muscle Wasting Ipsilateral to Symptoms in Patients with Acute/Subacute Low Back Pain. ***Spine***. 19:165-172.

- HIDES, J.A., RICHARDSON, C.A. & JULL, G.A. (1996). Multifidus Muscle Recovery is Not Automatic after Resolution of Acute, First-Episode Low Back Pain. *Spine*. 21: 2763-2769.
- HIEBERT, R., SKOVRON, M.L., NORDIN, M. & CRANE, M. (2003). Work Restrictions and Outcome of Nonspecific LBP. *Spine*. 28: 722-728.
- HILDEBRANDT, J.; URSIN, H.; MANNION, A.F.; AIRAKSINEN, O.; BROX, J.I.; KLABER-MOFFETT, J.; REIS, S.; ZANOLI, G.; CEDRASCHI, C.; KOVACS, F. AND STAAL, J.B. (2004). European Guidelines for the Management Chronic Non-Specific Low Back Pain *Cost B13 Working Group on Guidelines for Chronic Low Back Pain*
- HLOBIL, H., STAAL, J.B., TWISK, J., KÖKE, A., ARIËNS, G., SMID, T. & VAN MECHELEN, W. (2005). The Effects of a Graded Activity Intervention for Low Back Pain in Occupational Health on Sick Leave, Functional Status and Pain: 12-Month Results of a Randomized Controlled Trail. *Journal of Occupational Rehabilitation*. 15(4): 569-580.
- HODGES, P.W. & RICHARDSON, C.A. (1996). Inefficient Muscular Stabilization of the Lumbar Spine Associated with Low Back Pain: A Motor Control Evaluation of Transversus Abdominis. *Spine*. 21(22): 2640-2650.
- HODGES, P.W. & RICHARDSON, C.A. (1997). Feedforward Contraction of Transversus Abdominis is not Influenced by the Direction of Arm Movement. *Experimental Brain Research*. 114: 362-370.
- HODGES, P.W. & RICHARDSON, C.A. (1998). Delayed Postural Contraction of Transversus Abdominis in Low Back Pain Associated with Movement of the Lower Limb. *Journal of Spinal Disorders*. 11: 46-56.



- HODGES, P.W.&RICHARDSON, C.A. (1999). Altered Trunk Muscle Recruitment in People with Low Back Pain with Upper Limb Movement at Different Speeds. *Archives of Physical Medicine and Rehabilitation*. 80: 1005-1012.
- HODGES, P., CRESSWELL, A. & THORSTENSSON, A. (1999). Preparatory Trunk Motion Accompanies Rapid Upper Limb Movement. *Experimental Brain Research*. 124: 69-79.
- HODGES, P.W. (2001). Changes in Motor Planning of Feed-Forward Postural Responses of the Trunk Muscles in Low Back Pain. *Experimental Brain Research*. 141: 261-266.
- HODGES, P.W., CRESSWELL, A.G. & THORSTENSSON, A. (2001). Perturbed Upper Limb Movements cause Short-Latency Postural Responses in Trunk Muscles. *Experimental Brain Research*. 138: 243-250.
- HOLDCROFT, A. & JAGGER, S. (2005). **The Chronic Pain Patient** in **Core Topics in Pain**. London: CambridgeUniversity Press. P 117-120.
- HOLMES, J. MACD & SWORN, B.R. (1945). Sciatic Neuritis. *British Medical Journal*. 2: 350-351.
- HOOGENDOORN, W.E., VAN POPPEL, M.N.M., BONGERS, P.M., KOES, B.W. & BOUTER, L.M.(2000). Systematic Review of Psychosocial Factors at Work and in Private Life as Risk Factors for Back Pain. *Spine*. 25: 2114-2125.
- HORAK, F.B., ESSELMAN, P., ANDERSON, M.E.& LYNCH, M.K. (1984). The Effects of Movement Velocity, Mass Displacement, and Task Certainty on Associated

Postural Adjustments made by Normal and Hemiplegic Individuals. ***Journal of Neurology, Neurosurgery, and Psychiatry.***

HOYLAND, J.A., FREEMONT, A.J. & JAYSON, M.I.V. (1989). Intervertebral Foramen Venous Obstruction, a Cause of Periradicular Fibrosis. ***Spine.*** 14: 558-568.

HOYT, W.H., HUNT, H.H., DE PAUW, M.A., BARD, D., SHAFFER, F., PASSIAS, J.N., ROBBINS, D.H., RUNYON, D.G., SEMRAD, S.E., SYMONDS, J.T. & WATT, K.C. (1981). Electromyographic Assessment of Chronic Low Back Pain Syndrome. ***Journal of the American Osteopathic Association.*** 80: 57-59.

HUMPHREY, A.R., NARGOL, A.V., JONES, A.P., RATCLIFFE, A.A. & GREENOUGH, C.G. (2005). The Value of Electromyography of the Lumbar Paraspinal Muscles in Discriminating between Chronic Low Back Pain Sufferers and Normal Subjects. ***European Spine Journal.*** 14: 175-184.

HULTMAN, G., NORDIN, M., SARASTE, H. & OHLSEN, H. (1993). Body Composition, Endurance, Strength, Cross-Sectional Areas, and Density of MM Erector Spinae in Men with and without Low Back Pain. ***Journal of Spinal Disorders.*** 6: 114-123.

HURWITZ, E.L., MORGENSTERN, H., HARBER, P., KOMINSKI, G.F., BELIN, T.R., YU, F. & ADAMS, A.H. (2002). A Randomized Trial of Medical Care with and without Physical Therapy and Chiropractic Care with and without Physical Modalities for Patients with Low Back Pain: 6-Month Follow-Up Outcomes from the UCLA Low Back Pain Study. ***Spine.*** 27: 2193-2204.

HYUN, J.K., LEE, J.Y., LEE, S.J. & JEON, J.Y. (2007). Asymmetric Atrophy of Multifidus Muscle in Patients with Unilateral Lumbosacral Radiculopathy. ***Spine.*** 32(21): E598-E602.

- IGUCHI, T., KANEMURA, A., KASAHARA, K., KURIHARA, A., DOITA, M. & YOSHIYA, S. (2003): Age Distribution of Three Radiologic Factors for Lumbar Instability: Probable Aging Process of the Instability with Disc Degeneration. **Spine**. 28: 2628-2633.
- IHLEBAEK, C., HANSSON, T., LAERUM, E., BRAGE, S., ERIKSEN, H.R., HOLM, S.H., SVENDSRØD, R. & INDAHL, A. (2006). Prevalence of Low Back Pain and Sickness Absence: A “Borderline” Study in Norway and Sweden. **Scandinavian Journal of Public Health**. 5: 555-558.
- INDAHL, A., VELUND, L. & REIKERAAS, O. (1995). Good Prognosis for Low Back Pain when Left Untampered: A Randomized Clinical Trail. **Spine**. 20: 473-477.
- INDAHL, A, KAIGLE, A.M., REIKERAS, O. & HOLM, S.H. (1997). Interaction between the Porcine Lumbar Intervertebral Disc, Zygapophysial Joints, and Paraspinal Muscles. **Spine**. 22: 2834-2840.
- INDAHL, A., HALDORSEN E.H. HOLM, S., REIKERÅS, O. & URSIN, H. (1998). Five-Year Follow-Up Study of a Controlled Clinical Trail Using Light Mobilizing and an Informative Approach to Low Back Pain. **Spine**. 23(23): 2625-2630.
- INMAN, V.T. & SAUNDERS, J.B. (1942). The Clinico-Anatomical Aspects of the Lumbo-Sacral Region. **Radiology**. 38: 669-678.
- INOUE, H. (1981). Three-Dimensional Architecture of Lumbar Intervetebreal Discs. **Spine**. 6: 139-146.
- ISMAIEL, A.H.M.A. & PORTER, R.W. (1992). Effect of Straight Leg Raising on Blood Pressure. **Spine**. 17: 1117-1118.

ITO, T., SHIRADO, O., SUZUKI, H., TAKAHASHI, M., KANEDA, K. & STRAX, T.E. (1996). Lumbar Trunk Muscle Endurance Testing: An Inexpensive Alternative to a Machine for Evaluation. ***Archives of Physical Medicine and Rehabilitation***. 77: 75-79.

JACKSON, C.P. & BROWN, M.D. (1983). Is there a Role for Exercise in the Treatment of Patients with Low Back Pain? ***Clinical Orthopaedics***. 39-45.

JÄNIG, W., LEVINE, J.D. & MICHAELIS, M. (1996). Interactions of Sympathetic and Primary Afferent Neurons following Nerve Injury and Tissue Trauma. In: KUMAZAWA, T., KRUGER, L. & MIZUMURA, K. (Editors) ***The Polymodal Receptor: A Gateway to Pathological Pain. Progress in Brain Research***. Vol 113. Elsevier Science, Amsterdam: pp161-184.

JÄNIG, W. & BARON, R. (2003). Complex Regional Pain Syndrome: Mystery Explained? ***Lancet Neurology***. 2:687-697.

JANSEN, J.P., HOOZEMANS, M.J.M., VAN DER BEEK, A.J. & FRINGS-DRESEN, M.H.W. (2002). Evaluation of Ergonomic Adjustments of Catering Carts to Reduce External Pushing Forces. ***Applied Ergonomics***. 33(2): 117-128.

JOHANNSEN, F., REMVIG, L., KRYGER, P., BECK, P., WARMING, S., LYBECK, K., DREYER, V. & LARSEN, L.H. (1995). Exercises for Chronic Low Back Pain: A Clinical Trial. ***The Journal of Orthopaedic and Sports Physical Therapy***. 22(2): 52-59.

JOHNSON, M.A., POLGAR, J., WEIGHTMAN, D. & APPLETON, D. (1973). Data on the Distribution of Fibre Types in Thirty-Six Human Muscles. An Autopsy Study. ***Journal of the Neurological Sciences***. 18: 111-129.

JOHNSON, M.I. (1997). The Physiology of the Sensory Dimensions of Clinical Pain. **Physiotherapy**. 83(10): 526-536.

JONES, D.A., RUTHERFORD, O.M. & PARKER, D.F. (1989). Physiological Changes in Skeletal Muscle as a Result of Strength Training. **Quarterly Journal of Experimental Physiology**. 74: 233-256.

JÖNSSON, B. & STRÖMQVIST, B. (1995). The Straight Leg Raising Test and the Severity of Symptoms in Lumbar Disc Herniation: A Preoperative Evaluation. **Spine**. 20: 27-30.

JÖNSSON, B. & STRÖMQVIST, B. (1996). Neurological Signs in Lumbar Disc Herniation. **Acta Orthopaedica Scandinavica**. 67: 466-469.

JORANSON, D.E., GILSON, A.M., DAHL, J.L. & HADDOX, J.D. (2002). Pain Management, Controlled Substances, and State Medical Board Policy: A Decade of Change. **Journal of Pain and Symptom Management**. 23(2): 138-147.

JORGENSEN, K. & NICOLAISEN, T. (1986). Two Methods for Determining Trunk Extensor Endurance: A Comparative Study. **European Journal of Applied Physiology**. 55: 639-644.

JORGENSEN, K., NICHOLAISEN, T. & KATO, M. (1993). Muscle Fibre Distribution, Capillary Density and Enzymatic Activities in the Lumbar Paravertebral Muscles of Young Men: Significance for Isometric Endurance. **Spine**. 18: 1439-1450.

- JORGENSEN, K. (1997). Human Trunk Extensor Physiology and Ergonomics. ***Acta Physiologica Scandinavica. Supplementum.*** 637: 1-58.
- JOSEPH, J. & MCCOLL, I. (1961). Electromyography of Muscles of Posture: Posterior Vertebral Muscles in Males. ***Journal of Physiology (London).*** 157: 33-37.
- JOUSSET, N., FANELLO, S., BONTOUX, L., DUBUS, V., BILLABERT, C., VIELLE, B., ROQUELAURE, Y., PENNEAU-FONTBONNE, D & RICHARD, I. (2004). Effects of Functional Restoration versus Three Hours per Week Physical Therapy: A Randomized Control Study. ***Spine.*** 29(5): 487-493.
- JUKER, D., MCGILL, S.M. & KROPF, P. (1998). Quantitative Intramuscular Myoelectric Activity of Lumbar Portions of Psoas and the Abdominal Wall during Cycling. ***Journal of Applied Biomechanics.*** 14(4): 428-438.
- JULL, G.A.&RICHARDSON, C.A. (1994). Rehabilitation of Active Stabilization of the Lumbar Spine. In: TWOMEY, L.T. & TAYLOR, J.R. (Eds.). ***Physical Therapy of the Low Back.*** New York, NY: Churchill Livingstone.
- JUSTINS, D.M. (2005). ***Pain 2005 – An Updated Review.*** Seattle: IASP Press, p 80.
- KÄÄPÄ, E.H., FRANTSI, K., SAMA, S. & MALMIVAARA, A. (2006). Multidisciplinary Group Rehabilitation versus Individual Physiotherapy for Chronic Non-Specific Low Back Pain: A Randomized Trail. ***Spine.*** 31(4): 371-376.
- KADI, F. (2000). Adaptation of Human Skeletal Muscle to Training and Anabolic Steroids. ***Acta Physiologica Scandinavica. Supplementum.*** 646: 1-52.

KAIGLE, A.M., HOLM, S.H. & HANSSON, T.H. (1995). Experimental Instability in the Lumbar Spine. *Spine*. 20: 421-430.

KALIMO, H., RANTANEN, J., VILJANEN, T. & EINOLA, S. (1989). Lumbar Muscles: Structure and Function. *Annals of Medicine*. 21: 353-359.

KANAYAMA, M., ABUMI, K., KANEDA, K., TADANO, S. & UKAI, T. (1996). Phase Lag of the Intersegmental Motion in Flexion-Extension of the Lumbar and Lumbosacral Spine. An In Vivo Study. *Spine*. 21: 1416-1422.

KANKAANPÄÄ, M., TAIMELA, S., LAAKSONEN, D., HANNINEN, O. & AIRAKSINEN, O. (1998). Back and Hip Extensor Fatigability in Chronic Low Back Pain Patients and Controls. *Archives of Physical Medicine and Rehabilitation*. 79: 412-417.

KANKAANPÄÄ, M., TAIMELA, S., AIRAKSINEN, O. & HANNINEN, O. (1999). The Efficacy of Active Rehabilitation in Chronic Low Back Pain. Effect on Pain Intensity, Self-Experienced Disability, and Lumbar Fatigability. *Spine*. 24: 1034-1042.

KANKAANPÄÄ, M., COLIER, W.N., TAIMELA, S., ANDERS, C., AIRAKSINEN, O., KOKKO-ARO, S.M. & HÄNNINEN, O. (2005). Back Extensor Muscle Oxygenation and Fatigability in Healthy Subjects and in Low back Pain Patients during Dynamic Back Extension Exertion. *Pathophysiology*. 12: 267-273.

KARJALAINEN, K., MALMIVAARA, A., VAN TULDER, M., ROINE, R., JAUHAINEN, M., HURRI, H. & KOES, B.W. (2001). Multidisciplinary Biopsychosocial Rehabilitation for Neck and Shoulder Pain among Working Age Adults. *Spine*. 26: 174-181.

KARJALAINEN, K., MALMIVAARA, A., VAN TULDER, M., ROINE, R., JAUHAINEN, M., HURRI, H. & KOES, B.W. (2003a). Multidisciplinary Rehabilitation of Fibromyalgia

and Musculoskeletal Pain in Working Adults (Cochrane Review). **Cochrane Database System Review** (2): CD001984.

KARJALAINEN, K., MALMIVAARA, A., POHJOLAINEN, T., HURRI, H., MUTANEN, P., RISSANEN, P., PAHKAJARVI, H., LEVON, H., KARPOFF, H. & ROINE, R. (2003b). Mini-Intervention for Subacute Low Back Pain: A Randomized Controlled Trial. **Spine**. 28: 533-540.

KARST, G.M. & WILLETT, G.M. (2004). Effects of Specific Exercise Instructions on Abdominal Muscle Activity during Trunk Curl Exercises. **Journal of Orthopaedic & Sports Physical Therapy**. 34(1): 4-12.

KASER, L., MANNION, A.F., RHYNER, A., WEBER, E., DVORAK, J. & MUNTENER, M. (2001). Active Therapy for Chronic Low Back Pain: Part 2. Effects on Paraspinal Muscle Cross-Sectional Area, Fibre Type Size, and Distribution. **Spine**. 26: 909-919.

KATZ, L. (2000). The Multidimensional Nature of Chronic Pain: An Integrative Model for the Interdisciplinary Team. **CME**. 18(2): 139-143.

KATZ, R. (2006). Impairment and Disability Rating in Low Back Pain. **Clinics in Occupational and Environmental Medicine**. 5(3): 719-740.

KAUPILLA, L.I. (1995). In-Growth of Blood Vessels in Disc Degeneration Angiographic and Histological Studies of Cadaver Spines. **The Journal of Bone and Joint Surgery. American Volume**. 77: 26-31.



- KAUPPILA, L.I., MCALINDON, T., EVANS, S., WILSON, P.W., KIEL, D. & FELSON, D.T. (1997). Disc Degeneration / Back Pain and Calcification of the Abdominal Aorta: A 25-Year Follow-Up Study in Framingham. *Spine*. 22: 1642-1649.
- KAUPPILA, L.I., MIKKONEN, R., MANKINEN, P., PELTO-VASENIUS, K. & MAENPAA, I. (2004). MR Aortography and Serum Cholesterol Levels in Patients with Long-Term Nonspecific Lower Back Pain. *Spine*. 29: 2147-2152.
- KAVCIC, N., GRENIER, S. & MCGILL, S.M. (2004). Quantifying Tissue Loads and Spine Stability while Performing Commonly Prescribed Low Back Stabilizing Exercises. *Spine*. 20: 2319-2329.
- KEEFE, F.J., BROWN, G.K., WALLSTON, K.A. & CALDWELL, D.S. (1989). Coping with Rheumatoid Arthritis: Catastrophising as a Maladaptive Strategy. *Pain*. 37: 51-56.
- KEEFE, F.J., RUMBLE, M.E., SCIOPIO, C.D., GIORDANO, L.A. & PERRI, L.M. (2004). Psychological aspects of Persistent Pain: Current State of the Science. *The Journal of Pain: Official Journal of the American Pain Society*. 5: 195-211.
- KEEL, P.J., WITTIG, R., DEUTSCHMANN, R., DIETHELM, U., KNÜSEL, O., LÖSCHMANN, C., MATATHIA, R., RUDOLF, T. & SPRING, H. (1998). Effectiveness of Inpatient Rehabilitation for Subchronic and Chronic Low Back Pain by an Integrative Group Treatment Program (Swiss Multicentre Study). *Scandinavian Journal of Rehabilitation Medicine*. 30(4): 211-219.
- KELLER, A., BROX, J.I., GUNDERSON, R., HOLM, I., FRIIS, A. & REIKERÅS, O. (2003). Trunk Muscle Strength, Cross-Sectional Area, and Density in Patients with Chronic Low Back Pain Randomized to Lumbar Fusion or Cognitive Intervention and Exercise. *Spine*. 29(1): 3-8.

- KELSEY, J.L., GITHENS, P.B., WHITE, A.A. III., HOLFORD, T.R., WALTER, S.D., O'CONNOR, T., OSTFELD, A.M., WEIL, U., SOUTHWICK, W.O. & CALOGERO, J.A. (1984). An Epidemiological Study of Lifting and Twisting on the Job and Risk for Acute Prolapsed Lumbar Intervertebral Disc. ***Journal of Orthopaedic Research: Official Publication of the Orthopaedic Research Society***. 2(1): 61-66.
- KENDALL, F.P., MCCREARY, E.K. & PROVANCE, P.G. (1993). **Muscles Testing and Function with Posture and Pain**. (Fourth Edition). Philadelphia, USA: Lippincott Williams & Wilkins.
- KENDALL, N.A. (1999). Psychological Approaches to the Prevention of Chronic Pain: The Low Back Paradigm. ***Baillieres Best Practice & Research: Clinical Rheumatology***. 13: 545-554.
- KIKUCHI, S. (2008). New Concept for Backache: Biopsychosocial Pain Syndrome. ***European Spine Journal***. 17(4): S421-S427.
- KINGERY, W.S., GUO, T.Z., DAVIES, M.E., LIMBIRD, L. & MAZE, M. (2000). The Alpha (2A) Adrenoceptor and the Sympathetic Postganglionic Neuron Contribute to the Development of Neuropathic Heat Hyperalgesia in Mice. ***Pain***. 85: 345-358.
- KLENERMAN, L., SLADE, P.D., STANLEY, I.M., PENNIE, B., REILLY, J.P., ATCHINSON, L.E., TROUP, J.D. & ROSE, M.J. (1995). The Prediction of Chronicity in Patients with an Acute Attack of Low Back Pain in a General Practice Setting. ***Spine***. 20: 478-484.
- KNUTSSON, F. (1944). The Instability Associated with Disc Degeneration in the Lumbar Spine. ***Acta Radiologica Scandinavica***. 25: 593-609.

- KOBAYASHI, S., YOSHIKAWA, H., HACHIYA, Y., UKAI, T. & MORITA, T. (1993). Vasogenic Edema Induced by Compression Injury to the Spinal Nerve Root. *Spine*. 18: 1410-1424.
- KOES, B.W., BOUTER, L.M., BECKERMANN, H., VAN DER HEIJDEN, G.J.M.G. & KNIPSCHILD, P.G. (1991). Physiotherapy Exercises and Back Pain: A Blinded Review. *BMJ*. 302: 1572-1576.
- KOES, B.W., ASSENDELFT, W.J.J., VAN DER HEIJDEN, G.J.M.G. & BOUTER, L.M. (1996). Spinal Manipulations for Low Back Pain: An Updated Systematic Review of Randomized Control Trails. *Spine*. 21(24): 2860-2871.
- KOES, B.W., VAN TULDER, M.W., OSTELO, R., BURTON, K.A. & WADDELL, G.(2001). Clinical Guidelines for the Management of Low Back Pain in Primary Care. *Spine*. 26: 2504-2514.
- KOHLES, S., BARNES, D., GATCHEL, R.J. & MAYER, T.G. (1990). Improved Physical Performance Outcomes after Functional Restoration in Patients with Chronic Low Back Pain. Early versus Recent Training Results. *Spine*. 15: 1321-1324.
- KOMI, P.V. (1986). Training of Muscle Strength and Power: Interaction of Neuromotoric, Hypertrophic, and Mechanical Factors. *International Journal Of Sports Medicine*. 7(suppl 1): 10-15.
- KONTTINEN, Y.T., GRONBLAD, M., ANTTI-POIKA, I., SEITSALOS, S., SANTAVIRTA, S., HUKKANEN, M. & POLAK, J.M. (1990). Neuroimmunohistochemical Analysis of Peridiscal Nociceptive Neural Elements. *Spine*. 15: 383-386.

KOOL, J., DE BIE, R., OESCH, P., KNUSEL, O., VAN DEN BRANDT, P. & BACHMANN, S. (2004). Exercise Reduces Sick Leave in Patients with Non-Acute Non-Specific LBP: A Meta-Analysis. ***Journal of Rehabilitation Medicine***. 36: 49-62.

KORI, S.H., MILLER, R.P. & TODD, D.D. (1990) Kinesiophobia: A New View of Chronic Pain Behavior. ***Pain Management***35-43.

KRATHWOHL, D.R. (1993). **Methods of Educational and Social Science Research**. New York, NY: Longman

KRESS, M. & REEH, P.W. (1996). Chemical Excitation and Sensitization in Nociceptors. In: BELMONTE, C. & CERVERO, E. (Editors) **Neurobiology of Nociceptors**. Oxford, UK: OxfordUniversity Press: 258-297.

KRISMER, M. & VAN TULDER, M. (2007). Strategies for Prevention and Management of Musculoskeletal Conditions. Low Back Pain (Non-Specific). ***Best Practice & Research. Clinical Rheumatology***. 1: 77-91.

LAM, S.K., JULL, G. & TRELEAVEN, J. (1989). Lumbar Spine Kinesthesia in Patients with Low Back Pain. ***The Journal of Orthopaedic and Sports Physical Therapy***. 29: 294-299.

LATIKKA, P., BATTIE, M.C., VIDEMAN, T. & GIBBONS, L.E. (1995). Correlations of Isokinetic and Psychophysical Back Lift and Static Back Extensor Endurance Tests in Men. ***Clinical Biomechanics (Bristol, Avon)***. 10: 325-330.

LATIMER, J., MAHER, C.G., REFSHAUGE, K. & COLACO, I. (1999). The Reliability and Validity of the Biering-Sorensen Test in Asymptomatic Subjects and Subjects Reporting Current or Previous Non-Specific Low Back Pain. ***Spine***. 24: 2085-2090.

- LAURSEN, B. & SCIBYE, B. (2002). The Effect of Different Surfaces on Biomechanical Loading of Shoulder and Lumbar Spine during Pushing and Pulling of Two-Wheeled Containers. *Applied Ergonomics*. 33(2): 167-174.
- LAVENDER, S.A., MIRKA, G.A., SCHOENMARKLIN-SOMMERICH, C.M., SUDHAKAR, L.R. & MARRAS, W.S. (1989). The Effects of Preview and Task Symmetry on Trunk Muscle Response to Sudden Loading. *Human Factors*. 31: 101-115.
- LAVENDER, S.A., TSUANG, Y.H., HAFEZI, A., ANDERSSON, G.B., CHAFFIN, D.B. & HUGHES, R.E. (1992). Coactivation of the Trunk Muscles during Asymmetric Loading of the Torso. *Human Factors*. 34: 239-247.
- LAVENDER, S.A., MARRAS, W.S. & MILLER, R.A. (1993). The Development of Response Strategies in Preparation for Sudden Loading to the Torso. *Spine*. 18: 2097-2105.
- LEBOEUF-YDE, C. (2004). Back Pain – Individual and Genetic Factors. *Journal of Electromyography and Kinesiology*. 14: 129-133.
- LEE, D.H., LIU, X., KIM, H.T., CHUNG, K. & CHUNG, J.M. (1999). Receptor Subtype Mediating the Adrenergic Sensitivity of Pain Behavior and Ectopic Discharges in Neuropathic Lewis Rats. *Journal of Neurophysiology*. 81: 2226-2233.
- LEHMAN, G. & MCGILL, S.M. (2001). Quantification of the Differences in EMG Magnitude between Upper and Lower Rectus Abdominis during Selected Trunk Exercises. *Physical Therapy*. 81: 1096-1101.

- LEINONEN, V., KANKAANPÄÄ. M., LUUKKONEN, M. HANNINEN, O., AIRAKSINEN, O. & TAIMELA, S.(2001). Disc Herniation-Related Back Pain Impairs Feed-Forward Control of Paraspinal Muscles. **Spine**. 26: E367-372.
- LEINONEN, V., MÄÄTTÄ, S., TAIMELA, S., HERNO, A., KANKAANPÄÄ, M., PARTANEN, J., KANSANEN, M., HÄNNINEN, O. & AIRAKSINEN, O.(2002). Impaired Lumbar Position Sense in Association with Postural Stability and Motor and Somatosensory Evoked Potential Findings in Lumbar Spinal Stenosis. **Spine**. 27: 975-983.
- LEINONEN, V., KANKAANPÄÄ. M., LUUKKONEN, M., KANSANEN, M., HÄNNINEN, O., AIRAKSINEN, O. & TAIMELA, S. (2003). Lumbar Paraspinal Muscle Function, Perception of Lumbar Position, and Postural Control in Disc Herniation-Related Back Pain. **Spine**. 28(8): 842-848.
- LETHEM, J., SLADE, P.D., TROUP, J.D. & BENTLEY, G. (1983). Outline of Fear-Avoidance Model of Exaggerated Pain Perception: I. **Behaviour Research and Therapy**. 21: 401-408.
- LEWIN, G.R. RUEFF, A. & MENDELL, L.M. (1994). Peripheral and Central Mechanisms of NGF-Induced Hyperalgesia. **European Journal of Neuroscience**. 6: 1903-1912.
- LEVY, D., TAL, M., HOKE, A. & ZOCHODNE, D.W. (2000). Transient Action of the Endothelial Constitutive Nitric Oxide Synthase (ecNOS) Mediates the Development of Thermal Hypersensitivity following Peripheral Nerve Injury. **European Journal of Neuroscience**. 12: 2323-2332.
- LIDDLE, S.D., BAXTER, G.D. & GRACEY, J.H. (2004). Exercise and Chronic Low Back Pain: What Works? **Pain**. 107: 176-190.

- LIEBER, R.L. (1992). **Skeletal Muscle Structure and Function: Implications for Rehabilitation and Sports Medicine**. Baltimore, ML: Williams & Wilkins.
- LIEBER, R.L. (2002). Skeletal Muscle Structure, Function and Plasticity. In **The Physiological Basis for Rehabilitation**. (2<sup>nd</sup> edition). Baltimore: Lippincott, Williams & Wilkins.
- LINDAHL, O. (1966). Determination of the Segmental Mobility of the Lumbar Spine. **Acta Orthopaedica Scandinavica**. 37: 241-254.
- LINDGREN, K., SIHVONEN, T., LEINO, E. & PITKANEN, M. (1993). Exercise Therapy Effects on Functional Radiographic Findings and Segmental Electromyographic Activity in Lumbar Spine Instability. **Archives of Physical Medicine and Rehabilitation**. 74: 933-939.
- LINDSTROM, I., OHLUND, C., EEK, C., WALLIN, L., PETERSON, L-E., FORDYCE, W.E. & NACHEMSON, A.L. (1992). The Effect of Graded Activity on Patients with Subacute Low Back Pain: A Randomized Prospective Clinical Study with an Operant-Conditioning Behavioural Approach. **Physical Therapy**. 72: 279-293.
- LINDSTROM, I., OHLUND, C., & NACHEMSON, A. (1995). Physical Performance, Pain, Pain Behavior and Subjective Disability in Patients with Subacute Low Back Pain. **Scandinavian Journal of Rehabilitation Medicine**. 27: 153-160.
- LINN, R.L. (1986). Quantitative Methods in Research on Teaching. In WITTROCK, M.C. (Ed.) **Handbook of Research on Teaching**. (3<sup>rd</sup> Edition). New York, NY: Macmillan

- LINTON, S. (1994). Chronic Back Pain: Integrating Psychological and Physical Therapy. ***Behavioral Medicine***. 20: 101-104.
- LINTON, S. (2000). A Review of Psychological Risk Factors in Back and Neck Pain. ***Spine***. 25: 1148-1156.
- LINTON, S. & ANDERSSON, T. (2000). Can Chronic Disability be Prevented: A Randomized Trial of a Cognitive-Behavioral Intervention and Two Forms of Information for Patients with Spinal Pain. ***Spine***25: 2825-2831.
- LINTON, S., BUER, N., VLAEYEN, J.W. & HELLSING, A. (2000). Are Fear-Avoidance Beliefs Related to the Inception of an Episode of Back Pain? A Prospective Study. ***Psychology & Health***. 14(6): 1051-1059.
- LINTON, S.J., BOERSMA, K., JANSSON, M., SVÄRD, L. & BOTVALDE, M. (2005). The Effect of Cognitive Behavioural and Physical Therapy Preventive Intervention on Pain-Related Sick Leave: A Randomized Control Trail. ***Clinical Journal of Pain***. 21: 109-119.
- LIU, C.N., MICHAELIS, M., AMIR, R. & DEVOR, M. (2000). Spinal Nerve Injury Enhances Subthreshold Membrane Potential Oscillations in DRG Neurons: Relation to Neuropathic Pain. ***Journal of Neurophysiology***. 84: 205-215.
- LJUNGGREN, A.E., WEBER, H., KOGSTAD, O., THOM, E. & KIRKESOLA, G.(1997). Effect of Exercise on Sick Leave due to Low Back Pain: A Randomized, Comparative, Long-Term Study. ***Spine***. 22: 1610-1616.



- LONG, D.M., BENDEBBA, M. & TORGERSON, W.S. (1996). Persistent Back Pain and Sciatica in the United States: Patients Characteristics. *Journal of Spinal Disorders*. 9: 40-58.
- LONEY, P.L. & STRATFORD, P.W. (1999). The Prevalence of Low Back Pain in Adults: A Methodological Review of the Literature. *Physical Therapy*. 79: 384-396.
- LOPEZ, A., MATHERS, C., EZZATI, M., JAMISON, D. & MURRAY, J. (2006). Global and Regional Burden of Disease and Risk Factors, 2001: Systematic Analysis of Population Health Data. *Lancet*. 367: 1747-1757.
- LOUDON, J.K, BELL, S.L. & JOHNSTON, J.M. (1998). *The Clinical Orthopedic Assessment Guide*. Champaign, IL: Human Kinetics.
- LOUW, Q.A., MORRIS, L.D. & GRIMMER-SOMERS, K. (2007). The Prevalence of Low Back Pain in Africa: A Systematic Review. *BMC Musculoskeletal Disorders*. 8(105): 1-14.
- LU, W.W., LUK, K.D.K., CHEUNG, K.M.C., FANG, D., HOLMES, A.D. & LEONG, J.C.Y. (2001). **Energy Absorption of Human Vertebral Body under Fatigue Loading**. In: Abstracts, International Society for Study of the Lumbar Spine, Edinburgh, Scotland, June 19-23.
- LUCAS, D. & BRESLER, B. (1961). Stability of the Ligamentous Spine. Tech Report No. 40, Biomechanics Laboratory, University of California, San Francisco.
- LUO, X., PIETROBON, R., SUN, S.X., LIU, G.G & HEY, L. (2004). Estimates and Patterns of Direct Health Care Expenditures among Individuals with Back Pain in the United States. *Spine*.29: 79-86.

- LUOTO, S., HELIOVAARA, M., HURRI, H. & ALARANTA, H. (1995). Static Back Endurance and the Risk of Low Back Pain. ***Clinical Biomechanics (Bristol, Avon)***. 10: 323-324.
- LUOTO, S., TAIMELA, S., HURRI, H., AALTO, H., PYYKKÖ, I. & ALARANTA, H.(1996). Psychomotor Speed and Postural Control in Chronic Low Back Pain Patients: A Controlled Follow-Up Study. ***Spine***. 21: 2621-2627.
- LYNN, B. (1996) Neurogenic Inflammation caused by Cutaneous Polymodal Receptors. ***Progress in Brain Research***. 113: 361-368.
- MACINTOSH, J.E. & BOGDUK, N. (1986). The Biomechanics of the Lumbar Multifidus. ***Clinical Biomechanics***. 1: 205-213.
- MACINTOSH, J.E., VALENCIA, F., BOGDUK, N. & MUNRO, R.R.(1986). The Morphology of the Human Lumbar Multifidus. ***Clinical Biomechanics***. 1(4): 196-204.
- MACINTOSH, J.E., BOGDUK, N. & GRACOVETSKY, S. (1987). The Biomechanics of the Thoracolumbar Fascia. ***Clinical Biomechanics***. 2: 78-83.
- MAFFEY-WARD, L., JULL, G. & WELLINGTON, L. (1996). Towards a Clinical Test of Lumbar Spine Kinesthesia. ***The Journal of Orthopaedic and Sports Physical Therapy***. 24: 354-358.
- MAGNUSSON, M.L., ALEKSIEV, A., WILDER, D.G., POPE, M.H., SPRATT, K.F., LEE, S.H., GOEL, V.K. & WEINSTEIN, J.N.(1996). Unexpected Load and Asymmetric Posture as Etiologic Factors in Low Back Pain. ***European Spine Journal***. 5: 23-35.

MAGNUSSON, M.L., BISHOP, J.B., HASSELQUIST, L., SPRATT, K.F. & SZPALSKI, M. (1998). Range of Motion and Motion Patterns in Patients with Low Back Pain before and After Rehabilitation. *Spine*. 23:2631-2639.

MAHER, C., LATIMER, J. & REFSHAUGE, K. (1999). Prescription of Activities for Low Back Pain: What Works? *The Australian Journal of Physiotherapy*. 45: 121-132.

MACFARLANE, G.J., THOMAS, E., CROFT, P.R., PAPAGEORGIOU, A.C., JAYSON, M.I.V. & SILMAN, A.J. (1999). Predictors of Early Improvement in Low Back Pain amongst Consultants to General Practice: The Influence of Pre-Morbid and Episode-Related Factors. *Pain*. 80: 113-119.

MAIN, C.J. & WATSON, P.J. (1999). Psychological Aspects of Pain. *Manual Therapy*. 4: 203-215.

MAIN, C.J. & WILLIAM, A. (2002). ABC of Psychological Medicine: Muskulo-Skeletal Pain. *British Medical Journal*. 325: 534-537.

MAJLESI, J., TOGAY, H., UNALAN, U. & TOPRAK, S. (2008). The Sensitivity and Specificity of the Slump and Straight Leg Raising Tests in Patients with Lumbar Disc Herniation. *Journal of Clinical Rheumatology*. 14(2): 87-91.

MANNICHE, C., HESSELSØE, G., BENTZEN, L., CHRISTENSEN, I. & LUNDBERG, E. (1988). Clinical Trial of Intensive Muscle Training for Chronic Low Back Pain. *Lancet*. 2: 1473-1476.

MANNICHE, C., LUNDBERG, E., CHRISTENSEN, I., BENTZEN, L. & HESSELSON, G. (1991). Intensive Dynamic Back Exercises for Chronic Low Back Pain: A Clinical Trial. *Pain*. 47: 53-63.

MANNICHE, C., ASMUSSEN, K., LAURITSEN, B., VINTERBERG, H., KARBO, H., ABILDSTRUP, S., FISCHER-NIELSEN, K., KREBS, R. & IBSEN, K. (1993). Intensive Dynamic Back Exercise with or without Hyperextension in Chronic Back Pain after Surgery for Lumbar Disc Protrusion: A Clinical Trial. *Spine*. 18: 560-567.

MANNION, A.F. & DOLAN, P. (1994). Electromyographic Median Frequency Changes during Isometric Contraction of the Back Extensors to Fatigue. *Spine*. 19: 1223-1229.

MANNION, A.F., CONNOLLY, B., WOOD, K. & DOLAN, P. (1997). The Use of Surface EMG Power Spectral Analysis in the Evaluation of Back Muscle Function. *Journal of Rehabilitation Research and Development*. 34: 427-439.

MANNION, A.F. (1999). Fibre Type Characteristics and Function of the Human Paraspinal Muscles: Normal Values and Changes in Association with Low Back Pain. *Journal of Electromyography and Kinesiology*. 9: 363-377.

MANNION, A.F., MUNTENER, M., TAIMELA, S. (1999). A Randomized Clinical Trial of Three Active Therapies for Chronic Low Back Pain. *Spine*. 24: 2435-2448.

MANNION, A.F., KASER, L., WEBER, E., RHYNER, A., DVORAK, J. & MUNTENER, M. (2000). Influence of Age and Duration of Symptoms on Fibre Type Distribution and Size of the Back Muscles in Chronic Low Back Pain Patients. *European Spine Journal*. 9: 273-281.

- MANNION, A.F., TAIMELA, S., MUNTENER, M. & DVORAK, J. (2001a). Active Therapy for Chronic Low Back Pain: Part I. Effects on Back Muscle Activation, Fatigability, and Strength. *Spine*. 26: 897-908.
- MANNION, A.F., JUNGE, A., TAIMELA, S., MUNTENER, M., LORENZO, K. & DVORAK, J. (2001b). Active Therapy for Chronic Low Back Pain: Part 3. Factors Influencing Self-Related Disability and its Change following Therapy. *Spine*. 26: 920-929.
- MARCHAND, F. & AHMED, A.M. (1990). Investigation of the Laminate Structure of the Lumbar Disc Anulus Fibrosis. *Spine*. 15: 402-410.
- MARRAS, W.S., RANGARAJULU, S.L. & LAVENDER, S.A. (1987). Trunk Loading and Expectation. *Ergonomics*. 30: 551-562.
- MARRAS, W.S. & MIRKA, G.A. (1993). Electromyographic Studies of the Lumbar Trunk Musculature during the Generation of Low-Level Trunk Acceleration. *Journal of Orthopaedic Research: Official Publication of the Orthopaedic Research Society*. 11: 811-817.
- MARRAS, W.S., LAVENDER, S.A., LEURGANS, S., FATHALLAH, F.A., FERGUSON, S.A., ALLREAD, W.G. & RAJULU, S.L.(1995a). Biomechanical Risk Factors for Occupationally Related Low Back Disorder Risk. *Ergonomics*. 38: 377-410.
- MARRAS, W.S., PARNIANPOUR, M., FERGUSON, S.A., KIM, J.Y., GROWELL, R.R., BOSE, S. & SIMON, S.R. (1995b). The Classification of Anatomic- and Symptom-Based Low Back Disorders Using Motion Measure Models. *Spine*. 20:2531-2546.
- MARRAS, W.S., ALLREAD, W.G., BURR, D.L. & FATALLAH, F.A. (2000). Prospective Validation of a Low Back Disorder Risk Model and Assessment of Ergonomic

Interventions Associated with Manual Materials Handling Tasks. *Ergonomics*. 43: 1866-1886.

MARTIN, J.E., DUBBERT, P.H. & KATELL, A.D. (1984). Behavioral Control of Exercise in Sedentary Adults. Studies 1 through 6. *Journal of Consulting and Clinical Psychology*. 52: 795-811.

MARTINI, F.H. (1998). **Fundamentals of Anatomy & Physiology**. (4<sup>th</sup> Edition). New Jersey, NY: Prentice Hall.

MATTILA, M., HURME, M., ALARANTA, H., PALJÄRV, L., KALIMO, H., FALCK, B., LEHTO, M., EINOLA, S. & JÄRVINEN, M.(1986). The Multifidus Muscle in Patients with Lumbar Disc Herniation: A Histochemical and Morphometric Analysis of Intraoperative Biopsies. *Spine*. 11: 732-738.

MAY, A. (2008). Chronic Pain May Change the Structure of the Brain. *Pain*. 137: 7-15.

MAYER, T.G., GATCHEL, R.J., KISHINO, N., KEELEY, J., CAPRA, P.N., MAYER, H., BARNETT, J. & MOONEY, V. (1985). Objective Assessment of Spine Function following Industrial Injury: A Prospective Study with Comparison Group and One-Year Follow-Up. *Spine*. 10: 482-493.

MAYER, J.M., GRAVES, J.E., ROBERTSON, V.L., PIERRA, E.A., VERNA, J.L. & PLOUTZ-SNYDER, L.L. (1999). Electromyographic Activity of the Lumbar Extensor Muscles: Effect of Angle and Position during Roman Chair Exercise. *Archives of Physical Medicine and Rehabilitation*. 80: 751-755.

- MAYER, T., GATCHEL, R., BETANCUR, J. & Bovasso, E. (1995). Trunk Muscle Endurance Measurement: Isometric Contrasted to Isotonic Testing in Normal Subjects. ***Spine***. 20: 920-926.
- MAYER, T.G., POLATIN, P., SMITH, B., SMITH, C., GATCHEL, R., HERRING, S.A., HALL, H., DONELSON, R.G., DICKEY, J. & ENGLISH, W. (2001). Spine Rehabilitation: Secondary and Tertiary Nonoperative Care. In: ***Contemporary Concepts in Spine Care***. North American Spine Society. 1-11.
- MAYER, J.M., RALPH, L., LOOK, M., ERASALA, G.N., VERNA, J.L., MATHESON, L.N. & MOONEY, V. (2005). Treating Acute Low Back Pain with Continues Low-Level Heat Wrap Therapy and/or Exercise: A Randomized Controlled Trail. ***The Spine Journal***. 5: 395-403.
- MCCAFFERTY, W.B. & HORVATH, S.M. (1977). Specificity of Exercise and Specificity of Training: A Subcellular Review. ***Research Quarterly***. 48: 358-371.
- MCCARTNEY, N., HICKS, A.L., MARTIN, J. & WEBBER, C.E. (1995). Long-Term Resistance Training in the Elderly: Effects on Dynamic Strength, Exercise Capacity, Muscle, and Bone. ***The Journals of Gerontology. Series A, Biological Sciences and Medical Sciences***. 50: B97-B104.
- MCCARTHY, P.W., CARRUTHERS, B., MARTIN, D. & PETTS, P.(1991). Immunohistochemical Demonstration of Sensory Nerve Fibres and Endings in Lumbar Intervertebral Discs of the Rat. ***Spine***. 16: 653-655.
- MCCLESKEY, E.W. & GOLD, M.S. (1999). Ion Channels of Nociception. ***Annual Review of Physiology***. 61: 835-856.

- MCCOMBE, P.F., FAIRBANK, J.C.T., COCKERSOLE, B.C. & PYNSENT, P.B. (1989).  
Reproducibility of Physical Signs in Low Back Pain. *Spine*. 14: 908-917.
- MCGILL, S.M. & NORMAN, R.W. (1986). Partitioning of the L4-L5 Dynamic Moment into  
Disc, Ligamentous, and Muscular Components during Lifting. *Spine*. 11: 666-678.
- MCGILL, S.M. & NORMAN, R.W. (1987). Reassessment of the Role of Intra-Abdominal  
Pressure in Spinal Compression. *Ergonomics*. 30: 1565-1588.
- MCGILL, S.M., PATT, N. & NORMAN, R.W. (1988). Measurement of the Trunk Musculature  
of Active Males using CT Scan Radiography: Duplications for Force and Moment  
Generating Capacity about the L4/L5 Joint. *Journal of Biomechanics*. 21(4): 329.
- MCGILL, S.M. (1991). The Kinetic Potential of the Lumbar Trunk Musculature about Three  
Orthogonal Orthopaedic Axes in Extreme Postures. *Spine*. 16(7): 809-815.
- MCGILL, S.M. (1991). Kinetic Potential of the Lumbar Trunk Musculature about Three  
Orthogonal Orthopaedic Axes in Extreme Postures. *Spine*. 16: 809-815.
- MCGILL, S.M. (1992). A Myoelectrically Based Dynamic 3-D Model to Predict Loads on  
Lumbar Spine Tissues during Lateral Bending. *Journal of Biomechanics*. 25(4):  
395.
- MCGILL, S.M. & Brown, S. (1992). Creep Response of the Lumbar Spine to Prolonged  
Lumbar Flexion. *Clinical Biomechanics*. 7: 43.



- MCGILL, S.M. & NORMAN, R.W. (1993). Low Back Biomechanics in Industry: The Prevention of Injury through Safer Lifting. In: GRABINER, M.D. (Ed.). **Current Issues in Biomechanics**. Champaign, IL: Human Kinetics.
- MCGILL, S.M. (1997). The Biomechanics of Low Back Injury: Implications on Current Practice in Industry and the Clinic. **Journal of Biomechanics**. 30: 465-475.
- MCGILL, S.M. (1998). Low Back Exercises: Evidence for Improving Exercise Regiments. **Physical Therapy**. 78: 754-765.
- MCGILL, S.M., CHILDS, A. & LIEBENSON, C. (1999). Endurance Times for Stabilization Exercises: Clinical Targets for Testing and Training from a Normal Database. **Archives of Physical Medicine and Rehabilitation**. 80: 941-944.
- MCGILL, S.M. (2002). **Low Back Disorders: Evidence-Based Prevention and Rehabilitation**. Campaign, IL: Human Kinetics.
- MCINDOE, R. (1994). A Behavioral Approach to the Management of Chronic Pain. A Self-Management Perspective. **Australian Family Physician**. 23(12): 2284-2292.
- MCKENZIE, R.A. (1979). Prophylaxis in Recurrent Low Back Pain. **The New Zealand Medical Journal**. 89: 22.
- MCMAHON, M.J., GATCHEL, R.J., POLATIN, P.B. & MAYER, T.G. (1997). Early Childhood Abuse in Chronic Spinal Disorder Patients. A Major Barrier to Treatment Success. **Spine**. 22: 2408-2415.

- MEHRA, A., BAKER, D., DISNEY, S. & PYNSENT, P.B. (2008). Oswestry Disability Index Scoring Made Easy. *Annals of the Royal College of Surgeons of England*. 90: 497-499.
- MENS, J.M.A., VLEEMING, A., SNIJDERS, C.J., KOES, B.W. & STAM, H.J. (2001). Reliability and Validity of the Active Straight Leg Raise Test in Posterior Pelvic Pain since Pregnancy. *Spine*. 26(10): 1167-1171.
- MERSKEY, H., LOESER, J.D., DUBNER, R. & WADDELL, G. (2005). In: **The Paths of Pain 1975-2005**. Seattle: IASP Press: p 379-390.
- MELZACK, R. & WALL, P.D. (1965). Pain Mechanisms: A New Theory. *Science*. 150: 971-979.
- MELZACK, R. & WALL, P. (1988). **The Challenge of Pain**. London, UK: Penguin Books.
- MEYER, H.P. (2007). Pain Management in Primary Care – Current Perspectives. *South African Family Practice*. 49(7): 20-25.
- MICHAELIS, M., VOGEL, C., BLENK, K.H., ARNARSON, A. & JÄNIG, W. (1998). Inflammatory Mediators Sensitize Acutely Axotomized Nerve Fibres to Mechanical Stimulation in the Rat. *Journal of Neuroscience*. 18-7581-7587.
- MIOR, S. (2001). Exercise in the Treatment of Chronic Pain. *BMJ*. 322: 1511-1516.
- MITCHELL, R.I. & CARMEN, G.M. (1990). Results of a Multicenter Trial using an Intensive Active Exercise Program for the Treatment of Acute Soft Tissue and Back Injuries. *Spine*. 15: 514-521.

- MOFFROID, M.T., HAUGH, L.D., HAIG, A.J., HENRY, S.M. & POPE, M.H. (1993). Endurance Training of Trunk Extensor Muscles. *Physical Therapy*. 73: 3-10.
- MOFFROID, M.T, REID, S., HENRY, S.M., HAUGH, L.D. & RICAMATO, A. (1994). Some Endurance Measures in Persons with Chronic Low Back Pain. *The Journal of Orthopaedic and Sports Physical Therapy*. 20: 81-87.
- MOFFROID, M.T. (1997). Endurance of Trunk Muscles in Persons with Chronic Low Back Pain: Assessment, Performance, Training. *Journal of Rehabilitation Research and Development*. 34: 440-447.
- MOON, D.E., LEE, D.H., HAN, H.C. XIE, J., COGGESHALL, R.E. & CHUNG, J.M. (1999). Adrenergic Sensitivity of the Sensory Receptors Modulating Mechanical Allodynia in a Rat Neuropathic Pain Model. *Pain*. 80: 589-595.
- MOORE, J.E., VON KORFF, M., CHERKIN, D., SAUNDERS, K. & LORIG, K. (2000). A Randomized Trial of a Cognitive-Behavioral Program for Enhancing Back Pain Self Care in a Primary Setting. *Pain*. 88: 145-153.
- MOREAU, C.E., GREEN, B.N., JOHNSON, C.D. & MOREAU, S.R. (2001). Isometric Back Extension Endurance Tests: A Review of the Literature. *Journal of Manipulative and Physiological Therapeutics*. 24(2): 110-122.
- MORGAN, F.P. & KING, T. (1957). Primary Instability of Lumbar Vertebrae as a Common Cause of Low Back Pain. *The Journal of Bone and Joint Surgery. British Volume*.39: 6-22.

- MORELAND, J., FINCH, E., STRATFORD, P., BALSOR, B. & GILL, C. (1997). Interrater Reliability of Six Tests of Trunk Muscle Function and Endurance. ***The Journal of Orthopaedic and Sports Physical Therapy***. 26: 200-208.
- MORITANI, T., ODDSSON, L. & THORSTENSSON, A. (1991). Activation Patterns of the Soleus and Gastrocnemius Muscles during Different Motor Tasks. ***Journal of Electromyography and Kinesiology***. 1: 81-88.
- MORRIS, J., BRENNER, F. & LUCAS, D. (1962). An Electromyographic Study of the Intrinsic Muscles of the Back in Man. ***Journal of Anatomy***. 96: 509-520.
- MOSELEY, G.L., HODGES, P.W. & GANDEVIA, S.C. (2002). Deep and Superficial Fibers of the Lumbar Multifidus Muscle are Differentially Active during Voluntary Arm Movements. ***Spine***. 27(2): E29-E36.
- NACHEMSON, A.L. (1960). Lumbar Interdiscal Pressure. ***Acta Orthopaedica Scandinavica***. (suppl 43).
- NACHEMSON, A. & LINDH, M. (1969). Measurement of Abdominal and Back Extension Strength with and without Low Back Pain. ***Scandinavian Journal of Rehabilitation Medicine***. 1: 60-65.
- NACHEMSON, A., SCHULTZ, A.B. & BERKSON, M.H. (1979). Mechanical Properties of Human Lumbar Spine Motion Segments. Influence of Age, Sex, Disc Level, and Degeneration. ***Spine***. 4: 1-8.
- NACHEMSON, A. (1992). Newest Knowledge of Low Back Pain. ***La Clinica Ortopedica***. 279: 8-20.

- NELEMANS, P.J., DEBIE, R.A., DE VET, H.C. & STURMANS, F. (2001). Injection Therapy for Sub-Acute and Chronic LBP. **Spine**. 26: 501-515.
- NELSON, B.W., O'REILLY, E., MILLER, M., HOGAN, M., WEGNER, J.A. & KELLY, C. (1995). Focus on the Spine. The Clinical Effects of Intensive, Specific Exercise on Chronic Low Back Pain: A Controlled Study of 895 Consecutive Patients with 1-Year Follow-Up. **Orthopedics**. 18(10): 971-981.
- NEUGEBAUER, V. & SCHAIBLE, H.G. (1990). Evidence for a Central Component in the Sensitization of Spinal Neurons with Joint Input during Development of Acute Arthritis in Cat's Knee. **Journal of Neurophysiology**. 64: 299-311.
- NG, J.K. & RICHARDSON, C.A. (1996). Reliability of Electromyographic Power Spectral Analysis of Back Muscle Endurance in Healthy Subjects. **Archives of Physical Medicine and Rehabilitation**. 77: 259-264.
- NG, J.K., RICHARDSON, C.A. & JULL, G.A. (1997). Electromyographic Amplitude and Frequency Changes in the Iliocostalis Lumborum and Multifidus Muscles during a Trunk Loading Test. **Physical Therapy**. 77: 954-961.
- NG, J.K., RICHARDSON, C.A., KIPPERS, V. & PARNIANPOUR, M. (1998). Relationship between Muscle Fibre Composition and Functional Capacity of Back Muscles in Healthy Subjects and Patients with Back Pain. **The Journal of Orthopaedic and Sports Physical Therapy**. 27: 389-402.
- NIELSON, L.A. & NIELSON, T.G. (2003) Central Sensitization in Fibromyalgia and other Musculoskeletal Disorders. **Current Pain and Headache Reports**. 7: 355-361.

- NITZ, A.J. & PECK, D. (1986). Comparison of Muscle Spindle Concentrations in Large and Small Human Epaxial Muscles Acting in Parallel Combinations. ***American Surgeon***. 52: 273-277.
- NIV, D. & DEVOR, M. (2007). Position Paper of the European Federation of IASP Chapters (EFIC) on the Subject of Pain Management. ***European Journal of Pain***. 11: 487-489.
- NORDIN, M. & CAMPELLO, M. (1999). Physical Therapy: Exercises and the Modalities: When, What, and Why? ***Neurologic Clinics***. 17: 75-89.
- NOVY, D.M., SIMMONDS, M.J., OLSON, S.L., LEE, C.E. & JONES, S.C. (1999). Physical Performance: Differences in Men and Women with and Without Low Back Pain. ***Archives of Physical Medicine and Rehabilitation***. 80: 195-198.
- NUTTER, P. (1988). Occupational Exercise in the Treatment and Prevention of Low Back Pain. ***Occupational Medicine***. 3: 137-145.
- NUVUAGA, F. & NWUGA, V. (1985). Relative Therapeutic Efficiency of the Williams and McKenzie Protocols in Back Pain Management. ***Physiotherapy Practice***. 1: 99-105.
- NYKÄNEN, M. & KOIVISTO, K. (2004). Individual or Group Rehabilitation for People with Low Back Pain: A Comparative Study with 6-Month Follow-Up. ***Journal of Rehabilitation Medicine***. 36: 262-266.
- OCARINO, J.M., FONSECA, S.T., SILVA, P.L., MANCINI, M.C. & GONCALVES, G.G. (2008). Alternations of Stiffness and Resting Positions of the Elbow Joint Following Flexors Resistance Training. ***Manual Therapy***. 13(5): 411-418.

- O'CONNELL, J.E.A. (1943). Sciatica and the Mechanism of the Production of the Clinical Syndrome in Protrusions of the Lumbar Intervertebral Discs. *The British Journal of Surgery*. 30: 315-327.
- ODDSSON, L. & THORSTENSSON, A. (1990). Task Specificity in the Control of Intrinsic Trunk Muscles in Man. *Acta Physiologica Scandinavica*. 139: 123-131.
- OGILVIE, B.C. (1968). Psychological Consistencies within the Personality of High-Level Competitors. *JAMA: The Journal of the American Medical Association*. 205: 780-786.
- OGATA, K. (2002). **Modern Control Engineering**. (4<sup>th</sup> edition). Upper Saddle River, NJ: Prentice Hall.
- OLDERVOLL, L.M., RØ, M., ZWART, J.A. & SVEBAK, S. (2001). Comparison of Two Physical Exercise Programs for the Early Intervention of Pain in the Neck, Shoulder and Lower Back in Female Hospital Staff. *Journal of Rehabilitation Medicine*. 33: 156-161.
- OLDRIDGE, N.B. & STOLL, J.E. (1997). Spinal Disorders and Low Back Pain. In: SKINNER, J.S. (Editor). **Exercise Testing and Exercise Prescription for Special Cases**. (2<sup>nd</sup> Edition). Philadelphia, PA: Lea & Febiger.
- OMINO, K. & HAYASHI, Y. (1992). Preparation of Dynamic Posture and Occurrence of Low Back Pain. *Ergonomics*. 35: 693-707.

OMOKHODION, F. & SANYA, A. (2003): Risk Factors for Low Back Pain among Office Workers in Ibadan, Southwest Nigeria. ***Occupational Medicine***. 53: 287-289.

OMOKHODION, F. (2004). Low Back Pain in an Urban Population in Southwest Nigeria. ***Tropical Doctor***. 34: 17-20.

ÖRTENGREN, R., ANDERSSON, G.B. & NACHEMSON, A. (1981), Studies of Relationships between Lumbar Disc Pressure, Myoelectric Back Activity, and Intra-Abdominal (Intra-Gastric) Pressure. ***Spine***. 6: 98-103.

OSTELO, R.W.J.G., DE VET, H.C.W., WADDELL, G., KERCKHOFFS, M.R., LEFFERS, P. & VAN TULDER, M. (2003). Rehabilitation Following First-Time Lumbar Disc Surgery. ***Spine***. 28(3): 209-218.

O'SULLIVAN, P.B., PHYTY, G.D., TWOMEY, L.T. & ALLISON, G.T. (1997). Evaluation of Specific Stabilizing Exercises in the Treatment of Chronic Low Back Pain with Radiological Diagnosis of Spondylolysis or Spondylolisthesis. ***Spine***. 22: 2959-2967.

O'SULLIVAN, P.B., TWOMEY, L. & ALLISON, G.T. (1998). Altered Abdominal Muscle Recruitment in Patients with Chronic Back Pain Following a Specific Exercise Intervention. ***The Journal of Orthopaedic and Sports Physical Therapy***. 27: 114-124.

O'SULLIVAN, P. (2000). Lumbar Segmental 'Instability': Clinical Presentation and Specific Stabilizing Exercise Management. ***Manual Therapy***. 5: 2-12.



O'SULLIVAN, P.B., BURNETT, A., FLOYD, A.N., GADSDON, K., LOGIUDICE, J., MILLER, D. & QUIRKE, H. (2002). Lumbar Repositioning Deficit in a Specific Low Back Pain Population. **Spine**. 28(10): 1074-1079.

O'SULLIVAN, P.B., BURNETT, A., FLOYD, A.N., GADSDON, K., LOGIUDICE, J., MILLER, D. & QUIRKE, H. (2003). Lumbar Repositioning Deficit in a Specific Low Back Pain Population. **Spine**. 28(10): 1074-1079.

OKAWA, A., SHINOMIYA, K., KOMORI, H., MUNETA, T., ARAI, Y. & NAKAI, O.: Dynamic Motion Study of the Whole Lumbar Spine by Videofluoroscopy. **Spine**. 23: 1743-1749.

OSTELO, R.W.J.G. & DE VET, H.C.W. (2005). Clinically Important Outcomes in Low Back Pain. **Best Practice & Research: Clinical Rheumatology**. 19(4): 593-607.

OXLAND, T.R. & PANJABI, M.M. (1992). The Onset and Progression of Spinal Injury: A Demonstration of Neutral Zone Sensitivity. **Journal of Biomechanics**. 25: 1165-1172.

PALMGREN, T., GRÖNBLAD, M., VIRRI, J., KÄÄPÄ, E. & KARAHARJU, E. (1999). An Immunohistochemical Study of Nerve Structure in the Annulus Fibrosis of Human Normal Lumbar Intervertebral Discs. **Spine**. 24(20): 2075-2079.

PANJABI, M.M., ABUMI, K., DURANCEAU, J. & OXLAND, T. (1989). Spinal Stability and Intersegmental Muscle Forces: A Biomechanical Model. **Spine**. 14: 194-200.

PANJABI, M.M. (1992). The Stabilizing System of the Spine. Part II. Neutral Zone and Instability Hypothesis. **Journal of Spinal Disorders**. 5: 390-397.

PARKKOLA, R., KUJALA, U. & RYTOKOSKI, U. (1992). Response of the Trunk Muscles to Training Assessed by Magnetic Resonance Imaging and Muscle Strength. ***European Journal of Applied Physiology and Occupational Physiology***. 65: 383-387.

PARKKOLA, R., ALANEN, A., KALIMO, H., LILLSUNDE, I., KOMU, M. & KORMANO, M. (1993a). MR Relaxation Times and Fibre Type Predominance of the Psoas and Multifidus Muscle: An Autopsy Study. ***Acta Radiologica***. 34: 16-19.

PARKKOLA, R., RYTOKOSKI, U., KORMANO, M. (1993b). Magnetic Resonance Imaging of the Discs and Trunk Muscles in Patients with Chronic Low Back Pain and Healthy Control Subjects. ***Spine***. 18: 830-836.

PATTON, M.Q. (1987). ***How to Use Qualitative Methods in Evaluation***. Newbury Park, CA: Sage

PAULY, J.E. (1966). An Electromyographic Analysis of Certain Movement and Exercise: Some Deep Muscles of the Back. ***The Anatomical Record***. 155: 223-234.

PEARCY, M., PORTEK, I. & SHEPHERD, J. (1985). The Effect of Low Back Pain on Lumbar Spinal Movements Measured by Three-Dimensional X-Ray Analysis. ***Spine***. 10: 150-153.

PENGEL, L.H., HERBERT, R.D., MAHER, C.G. & REFSHAUGE, K.M. (2003). Acute LBP: Systematic Review of its Prognosis. ***BMJ***. 327: 323.

PENNYBACKER, J. (1940). Sciatica and the Intervertebral Disc. ***Lancet***. 238: 771-777.

- PERKINS, N.M.& TRACEY, D.J. (2000). Hyper Algesia due to Nerve Injury: Role of Neutrophils. ***Neuroscience***. 101: 745-757.
- PERKINS, J. & ZIPPLE, J.T. (2003). Nonspecific Low Back Pain. In: EHRMAN, J.K., GORDON, P.M., VISICH, P.S. & KETEYIAN, S.J. (Editors). **Clinical Exercise Physiology**. Champaign, IL: Human Kinetics.
- PETERKA, R.J. (2003). Simplifying the Complexities of Maintaining Balance. ***IEEE Engineering in Medicine and Biology Magazine: The Quarterly Magazine of the Engineering in Medicine & Biology Society***. 22: 63-68.
- PETERSEN, T., KRYGER, P., EKDAHL, C., OLSEN, S. & JACOBSEN, S. (2002). The Effect of McKenzie Therapy as Compared with that of Intensive Strengthening Training for the Treatment of Patients with Subacute or Chronic Low Back Pain: A Randomized Controlled Trail. ***Spine***. 27(16): 1702-1709.
- PFINGSTEN, M., HILDEBRANDT, J., SAUR, P., FRANZ, C. & SEEGER, D. (1997). Multidisciplinary Treatment Program on Chronic Low Back Pain, Part 4. Prognosis of Treatment Outcome and Final Conclusions. ***Schmerz***. 11: 30-41.
- PHELPS, G.L., VOGEL, R. & SHELLENBERGER, S. (2004). Treatment and Outcomes in Occupational Low Back Pain: A Practice Evaluation and Comparison with National and International Guidelines. ***Journal of Agromedicine***. 7: 67–78.
- PICAVET, H.S., VLAHEYEN, J.W. & SCHOUTEN, J.S. (2002). Pain Catastrophizing and Kinesiophobia: Predictors of Chronic Low Back Pain. ***American Journal of Epidemiology***. 156: 1028-1034.

- PINCUS, T., BURTON, A.K., VOGEL, S. & FIELD, A.P. (2002). A Systematic Review of Psychological Factors as Predictors of Chronicity/Disability in Prospective Cohorts of Low Back Pain. *Spine*. 27(5): E109-E120.
- PLAMONDON, A., SERRESSE, O., BOYD, K., LADOUCEUR, D. & DESJARDINS, P. (2002). Estimated Amounts at L5/S1 Level and Muscular Activation of Back Extensors for Six Prone Back Extension Exercises in Healthy Individuals. *Scandinavian Journal of Medicine & Science In Sports*. 12: 81-89.
- POLGER, J., JOHNSON, M.A., WEIGHTMAN, D. & APPLETON, D. (1973). Data on Fibre Size in Thirty-Six Human Muscles. *Journal of the Neurological Sciences*. 19: 307-318.
- PORTERFIELD, J.A. & DEROSA, C. (1998). **Mechanical Low Back Pain: Perspective in Functional Anatomy**. Philadelphia, PA: W.B. Saunders.
- PRADO-LEON, L., CELIS, A. & AVILA-CHAURAND, R. (2005). Occupational Lifting Tasks as a Risk Factor in Low Back Pain: A Case-Control Study in a Mexican Population. *Work*. 25: 107-114.
- PRENTICE, W.E. (2004). **Rehabilitation Techniques for Sport Medicine and Athletic Training**. (4<sup>th</sup> edition). Burr Ridge, IL: McGraw-Hill.
- PRITHVI RAJ, P. (2008). Intervertebral Disc: Anatomy-Physiology-Pathophysiology-Treatment. *Pain Practice*. 8(1): 18-44.
- PUNNETT, L., FINE, L.J., KEYERSERLING, W.M., HERRIN, G.D. & CHAFFIN, D.A. (1991). Back Disorders and Non-Neutral Trunk Postures of Automobile Assembly Workers. *Scandinavian Journal of Work Environment and Health*. 17: 337-346.

- PUNNETT, L., PRÜSS-USTÜN, A., NELSON, D.I., FINGERHUT, M.A., LEIGH, J., TAK, S-W. & PHILLIPS, S. (2005). Estimating the Global Burden of Low Back Pain Attributed to Combined Occupational Exposures. *The American Journal of Industrial Medicine*.
- QUINT, U., WILKE, H.J., SHIRAZI, A.A., PARNIANPOR, M., LOER, F. & CLAES, L.E. (1998). Importance of the Intersegmental Trunk Muscle for the Stability of the Lumbar Spine. A Biomechanical Study In Vitro. *Spine*. 23: 1937-1945.
- RADEBOLD, A., CHOLEWICKI, J., PANJABI, .M.M. & PATEL, T. (2000). Muscle Response Pattern to Sudden Trunk Loading in Healthy Individuals and in Patients with Chronic Low Back Pain. *Spine*: 25(8). 947-954.
- RADEBOLD, A., CHOLEWICKI, J., POLZHOFFER, G.K. & GREENE, H.S.(2001). Impaired Postural Control of the Lumbar Spine is Associated with Delayed Muscle Response Times in Patients with Chronic Idiopathic Low Back Pain. *Spine*. 26: 724-730.
- RAGLIN, J.S., MORGAN, W.P. & LUCHSINGER, A.E. (1990). Mood and Self-Motivation in Successful and Unsuccessful Female Rowers. *Medicine and Science in Sports and Exercise*. 22: 849-853.
- RAINVILLE, J., SOBEL, J., HARTIGAN, C., MONLUX, G. & BEAN, J. (1997). Decreasing Disability in Chronic Back Pain through Aggressive Spine Rehabilitation. *Journal of Rehabilitation Research and Development*. 34: 383-393.
- RAINVILLE, J., CARLSON, N., POLATIN, P., GATCHEL, R.J. & INDAHL, A. (2000). Exploration of Physicians' Recommendations for Activities in Chronic LBP. *Spine*. 25: 2210-2217.

- RAMER, M.S., MURPHY, P.G., RICHARDSON, P.M. & BISBY, M.A. (1998). Spinal Nerve Lesion-Induced Mechanoallodynia and Adrenergic Sprouting in Sensory Ganglia are attenuated in Interleukin-6 Knockout Mice. *Pain*. 78: 115-121.
- RANTANEN, J., HURME, M., FALCK, B., ALARANTA, H., NYKVIST, F., LEHTO, M., EINOLA, S. & KALIMO, H.(1993). The Lumbar Multifidus Muscle Five Years after Surgery for a Lumbar Intervertebral Disc Herniation. *Spine*. 18: 568-574.
- RASMUSSEN-BARR, E., ÄNG, B., ARVIDSSON, I. & NILSSON-WIKMAR, L. (2009). Graded Exercise for Recurrent Low Back Pain. A Randomized, Controlled Trial with 6-, 12- and 36-Month Follow-Ups. *Spine*. 34(3): 221-228.
- REBAIN, R., DO, G., BAXTER, D. & MCDONOUGH, S. (2002). A Systematic Review of the Passive Straight Leg Raising Test as a Diagnostic Aid for Low Back Pain (1989 – 2000). *Spine*. 27(17): E388-E395.
- REID, S., HAZARD, R.G. & FENWICK, J.W. (1991). Isokinetic Trunk-Strength Deficits in People with and Without Low-Back Pain: A Comparative Study with Consideration of Effort. *Journal of Spinal Disorders*. 4: 68-72.
- REILLY, K., LOVEJOY, B., WILLIAMS, R. & ROTH, H. (1989). Differences between a Supervised and Independent Strength and Conditioning Program with Chronic Low Back Syndromes. *Journal of Occupational Medicine: Official Publication of the Industrial Medical Association*. 31(6): 547-550.
- REPANTI, M., KOROVISSIS, P.G., STAMATAKAKIS, M.V., SPASTRIS, P. & KOSTI, P. (1998). Evolution of Disc Degeneration in Lumbar Spine: A Comparative Histological Study between Herniated and Post-Mortem Disc Specimens. *Journal of Spinal Disorders*. 11(1): 41-45.

Research Methods, Knowledge base;

<http://www.socialresearchmethods.net/kb/design.php>

Research methods, Knowledge base;

<http://www.socialresearchmethods.net/kb/statdesc.php>

RHOADS, J.M. (1977). Overwork. ***JAMA: The Journal of the American Medical Association***. 237: 2615-2618.

RICHARDSON, C., JULL, G., HODGES, P. HIDES, J.(1999). **Therapeutic Exercises for Spinal Segmental Stabilization in Low Back Pain**. London, UK: Churchill-Livingstone.

RICHARDSON, C.A., SNIJDERS, C.J., HIDES, J.A., DAMEN, L., PAS, M.S. & STORM, J. (2002). The Relation between the Transversus Abdominis Muscles, Sacroiliac Joint Mechanics, and Low Back Pain. ***Spine***. 27(4): 399-405.

RISSANEN, A., KALIMO, H. & ALARANTA, H. (1995). Effect of Intensive Training on the Isokinetic Strength and Structure of Lumbar Muscles in Patients with Chronic Low Back Pain. ***Spine***. 20: 333-340.

RIZZO, J.A., ABBOTT, T.A. & BERGER, M.L. (1998). The Labor Productivity Effects of Chronic Backache in the United States. ***Medical Care***. 36: 1471-1488.

ROBERTS, S., MENAGE, J. & URBAN, J.P.G. (1989). Biomechanical and Structural Properties of the Cartilage End-Plate and its Relationship to the Intervertebral Disc. ***Spine***. 14: 166.

- ROBERTS, S., EISENSTEIN, S.M., MENAGE, J., EVANS, E.H. & ASHTON, I.K. (1995). Mechanoreceptors in Intervertebral Discs. Morphology, Distribution and Neuropeptides. *Spine*. 20: 2645-2651.
- ROBERTS, S., EVANS, H., TRIVEDI, J. & MENAGE, J. (2006). Histology and Pathology of the Human Intervertebral Disc. *The Journal of Bone and Joint Surgery*. 88-A(2): 10-14.
- ROBINSON, J.L. & ROGERS, M.A. (1994). Adherence to Exercise Programs: Recommendations. *Sports Medicine*. 17: 39-52.
- ROME, L.C., FUNKE, R.P., ALEXANDER, R.M., LUTZ, G., ALDRIDGE, H., SCOTT, F. & FREADMAN, M. (1988). Why Animals have Different Muscle Fibre Types. *Nature*. 335: 824-827.
- ROME, J.D., TOWNSEND, C.O., BRUCE, B.K., SLETTEN, C.D., LUEDTKE, C.A. & HODGSON, J.E. (2004). Chronic Non-Cancer Pain Rehabilitation with Opioid withdrawal: Comparison of Treatment Outcomes Based on Opiate Status at Admission. *Mayo Clinic Proceedings. Mayo Clinic*. 79(6): 759-768.
- ROPPONEN, A., GIBBONS, L.E., VIDEMAN, T. & BATTIÉ, M.C. (2005). Isometric Back Extension Endurance Testing: Reasons for Test Termination. *Journal of Orthopaedic and Sports Physical Therapy*. 35: 437-442.
- ROTHWELL, J.C. (1994). Proprioceptors in Muscles, Joints and Skin. In: **Control of Human Voluntary Movement**. London, UK: Chapman & Hall.



- ROTHSTEIN, J.M. (1982). Muscle Biology. Clinical Considerations. *Physical Therapy*. 62: 1823-1830.
- SAAL, J.A. & SAAL, J.S. (1989). Non-Operative Treatment of Herniated Lumbar Intervertebral Disc with Radiculopathy: An Outcome Study. *Spine*. 14: 431-437.
- SAAL, J.A. (1990). Dynamic Muscular Stabilization in the Nonoperative Treatment of the Lumbar Pain Syndromes. *Orthopaedic Review*. 19: 691-700.
- SAAL, J.A. (1992). The New BackSchool Prescription: Stabilization Training Part II. *Occupation Medicine*. 7: 33-42.
- SALMINEN, J.J., MAKI, P., OKSENEEN, A. & PENTTI, J. (1992). Spinal Mobility and Trunk Muscle Strength in 15-Year-Old Schoolchildren with and without Low Back Pain. *Spine*. 17: 405-411.
- SANDERS, S.H. & BRENA, S.F. (1993). Empirically Derived Chronic Pain Patient Subgroups: The Utility of Multidimensional Clustering to Identify Different Treatment Effects. *Pain*. 54: 51-56.
- SANDERS, S.H., HARDEN, R.N., BENSON, S.E. & VICENTE, P.J. (1999). Clinical Practice Guidelines for Chronic Non-Malignant Pain Syndrome Patients II: An Evidence-Based Approach. *Journal of Back and Musculoskeletal Rehabilitation*. 13(2-3): 47-58.
- SANDERS, S.H., HARDEN, R.N. & VICENTE, P.J. (2005). Evidence-Based Clinical Practice Guidelines for Interdisciplinary Rehabilitation of Chronic Nonmalignant Pain Syndrome Patients. *Pain Practice*. 5(4): 303-315.

SANDKÜHLER, J. & LIU, X. (1998). Induction of Long-Term Potentiation at Spinal Synapses by Noxious Stimulation or Nerve Injury. *European Journal of Neuroscience*. 10: 2476-2480.

SAPA (2009). Counting the Cost of Back Pain. *Pretoria News, Tuesday March 10*.

SAUR, P., HILDEBRANDT, J., PFINGSTEN, M., SEEGER, D., STEINMETZ, U., STRAUB, A., HAHN, J., KASI, B., HEINEMANN, R. & KOCH, D. (1996). Multidisciplinary Treatment Program for Chronic Low Back Pain, Part 2. Somatic Aspects. *Der Schmerz*. 10(5): 237-253.

SCHAIBLE, H.G., SCHMIDT, R.F. & WILLIS, W.D. (1987). Enhancement of the Responses of Ascending Tract Cells in the Cat Spinal Cord by Acute Inflammation of the Knee Joint. *Experimental Brain Research*. 86: 489-499.

SCHAIBLE, H.G. & SCHMIDT, R.F. (1988). Time Course of Mechanosensitivity Changes in Articular Afferents during a Developing Experimental Arthritis. *Journal of Neurophysiology*. 60: 2180-2195.

SCHAIBLE, H.G. & SCHMIDT, R.F. (2000). Pathophysiologie von Nozizeption und Schmerz. In: FÖISCH, U.R., KOCHSIEK, K. & SCHMIDT, R.F. (Eds). **Pathophysiologie**. Berlin, Germany: Springer.

SCHAIBLE, H.G. (2004). The Neurophysiology of Pain. In: ISENBERG, D., MADDISON, P., WOO, P., GLASS, D. & BREEDVELD, F. (Eds). **The Oxford Textbook of Rheumatology**. (3<sup>rd</sup> edition). Oxford, UK: OxfordUniversity Press.

SCHAIBLE, H.G. & RICHTER, F. (2004). Pathophysiology of Pain. *Archives of Surgery*, 389: 237-243.

SCHMIDT, R.A. & LEE, T.D. (1998). **Motor Control and Learning: A Behavioral Emphasis**. Champaign, IL: Human Kinetics Publishers.

SCHMIDT-WILCKE, T., LEINISCH, E., GÄNSSBAUER, S., DRAGANSKI, B., BOGDAHN, U., ALTMEPPEN, J. & MAY, A. (2006). Affective Components and Intensity of Pain Correlated with Structural Difference in Gray Matter in Chronic Back Pain Patients. **Pain**. 125(1-2): 89-97.

SCHONSTEIN, E., KENNY, D., KEATING, J., KOES, B. & HERBERT, R.D. (2003). Physical Conditioning Programs for Workers with Back and Neck Pain: A Cochrane Systematic Review. **Spine**. 28: E391-E395.

SCHWARZER, A.C., APRILL, C.N., DERBY, R., FORTIN, J., KINE, G. & BOGDUK, N. (1994). The Relative Contributions of the Disc and Zygapophyseal Joint in Chronic Low Back Pain. **Spine**. 19: 801-806.

SEGOND VON BANCHET, G., PETROW, P.K., BRÄUER, R. & SCHAIBLE, H.G. (2000). Mono-Articular Antigen-Induced Arthritis Leads to Pronounced Bilateral Upregulation of the Expression of Neurokinin I and Bradykinin 2 Receptors in Dorsal Root Ganglion Neurons of Rats. **Arthritis Research**. 2: 424-427.

SEIFERT, F. & MAIHÖFNER, C. (2008). Central Mechanisms of Experimental and Chronic Neuropathic Pain: Findings from Functional Imaging Studies. **Cellular and Molecular Life Sciences**. 66(2009): 375-390.

SHERMAN, K.J., CHERKIN, D.C., ERRO, J., MIGLIORETTI, D.L. & DEYO, R.A. (2005). Comparing Yoga, Exercise and a Self-Care Book for Chronic Low Back Pain. **Annals of Internal Medicine**. 143: 849-856.

SHIPTON, E.A. (1999). **Pain: Acute and Chronic**. London, UK: Arnold.

SHIRADO, O., KANEDA, K. & ITO, T. (1992). Trunk-Muscle Strength during Concentric and Eccentric Contraction: A Comparison between Healthy Subjects and Patients with Chronic Low Back Pain. ***Journal of Spinal Disorders***. 5: 175-182.

SHIRADO, O., ITO, T., KANEDA, K. & STRAX, T.E.(1995a). Concentric and Eccentric Strength of Trunk Muscles: Influence of Test Postures on Strength and Characteristics of Patients with Chronic Low Back Pain. ***Archives of Physical Medicine and Rehabilitation***. 76(7): 604-611.

SHIRADO, O., ITO, T., KANEDA, K. & STRAX, T.E. (1995b). Electromyographic Analysis of Four Techniques for Isometric Trunk Muscle Exercise. ***Archives of Physical Medicine and Rehabilitation***. 76(3): 225-229.

SHIRADO, O., ITO, T., KIKUMOTO, T., TAKEDA, N., MINAMI, A. & STRAX, T.E. (2005). A Novel Back School Using a Multidisciplinary Team Approach Featuring Quantitative Functional Evaluation and Therapeutic Exercises for Patients with Chronic Low Back Pain. ***Spine***. 30(10): 1219-1225.

SIEBEN, J.M. VLAEYEN, J.W. TUERLINCKX, S. & PORTEQIJS, P.J. (2002). Pain-Related Fear in Acute Low Back Pain: The First Two Weeks of a New Episode. ***European Journal of Pain***. 6(3): 229-237.

SIMMONDS, M.J., OLSON, S.L., JONES, S., HUSSEIN, T., LEE, C.E., NOVY, D. & RADWAN, H. (1998). Psychometric Characteristics and Clinical Usefulness of Physical Performance Tests in Patients with Low Back Pain. ***Spine***. 23(22): 2412-2421.

- SIMMONDS, M.J. & DREISINGER, T.E. (2003). Low Back Pain Syndrome. In: DURSTINE, J.L. & MOORE, G.E. (2003). **ACSM's Exercise Management for Persons with Chronic Diseases and Disabilities**. Champaign, IL: Human Kinetics.
- SINCLAIR, S.J., HOGG-JOHNSON, S.H., MONDLOCH, M.V. & SHIELDS, S.A. (1997). The Effectiveness of an Early Active Intervention Program for Workers with Soft-Tissue Injuries. The Early Claimant Cohort Study. **Spine**. 22: 2919-2931.
- ŠIRCA, A. & KOSTEVC, V. (1985). The Fibre Type Composition of Thoracic and Lumbar Paravertebral Muscles in Man. **Journal of Anatomy**. 141: 131.
- SJOLIE, A.N. & LJUNGGREN, A.E. (2001). The Significance of High Lumbar Mobility and Low Lumbar Strength for Current and Future Low Back Pain in Adolescents. **Spine**. 26: 2629-2636.
- SLUIJS, E.M. & KNIBBE, J.J. (1991). Patient Compliance with Exercise: Different Theoretical Approaches to Short-Term and Long-Term Compliance. **Patient Education and Counseling**. 17: 191-204.
- SLUIJS, E.M., KOK, G.J. & VAN DER ZEE, J. (1993). Correlates of Exercise Compliance in Physical Therapy. **Physical Therapy**. 73: 771-786.
- SLUKA, K.A. & WALSH, D. (2003). Transcutaneous Electrical Nerve Stimulation: Basic Science Mechanism and Clinical Effectiveness. **The Journal of Pain: Official Journal of The American Pain Society**. 4: 109-121.
- SNIJDERS, C.J., RIBBERS, M.T.M., DE BAKKER, H.V., STOECKART, R. & STAM, H.J. (1998). EMG Recordings of Abdominal and Back Muscles in Various Standing

Postures: Validation of a Biomechanical Model on Sacroiliac Joint Stability. *Journal of Electromyography and Kinesiology: Official Journal of the International Society of Electrophysiological Kinesiology*. 8(4): 205-214.

SNOOK, S.H., WEBSTER, B.S., MCGORRY, R.W., FOGLEMAN, M.T. & MCCANN, K.B. (1998). The Reduction of Chronic Nonspecific Low Back Pain through the Control of Early Morning Lumbar Flexion: A Randomized Controlled Trail. *Spine*. 23(23): 2601-2607.

SODERBERG, G.L. & BARR, J.O. (1983). Muscular Function in Chronic Low Back Dysfunction. *Spine*. 8: 79-85.

SOLOMONOW, M., ZHOU, B., HARRIS, M., LU, Y. & BARATTA, R.V. (2000). The Ligamento-Muscular Stabilizing System of the Spine. *Spine*. 23: 2552-2562.

SOUKUP, M.G., GLOMSRÖL, B., LÖNN, J.H., BÖ, K. & LARSEN, S. (1999). The Effect of a Mensendieck Exercise Program as Secondary Prophylaxis for Recurrent LBP. A Randomized, Controlled Trail with 12-Month Follow-Up. *Spine*. 24: 1585-1591.

SPITZER, W., LEBLANC, F. & DUPUIS, M. (1987). Scientific Approach to the Assessment and Management of Activity-Related Spinal Disorders: A Monograph for Clinicians. Report of the Quebec Task Force on Spinal Disorders. *Spine*. 12(suppl): 1-59.

STAAL, J.B., HLOBIL, H., VAN TULDER, M.W., WADDELL, G., BURTON, A.K., KOES, B.W. & VAN MECHELEN, W. (2003). Occupational Health Guidelines for the Management of LBP: An International Comparison. *Occupational and Environmental Medicine*. 60: 618-626.

- STAAL, J.B. HLOBIL, H., TWISK, J.W., SMID, T., KÖKE, A.J. & VAN MECHELEN, W.(2004). Graded Activity for Low Back Pain in Occupational Health Care: A Randomized, Controlled Trial. ***Annals of Internal Medicine***. 140(2): 77-84.
- STAAL, J.B., RAINVILLE, J., FRITZ, J., VAN MECHELEN, W. & PRANSKY, G. (2005). Physical Exercise Interventions to Improve Disability and Return to Work in Low Back Pain: Current Insights and Opportunities for Improvement. ***Journal of Occupational Rehabilitation***. 15(4): 491-505.
- SLADE, S. & KEATING, J.L. (2006). Trunk Strengthening Exercises for Chronic Low Back Pain: A Systematic Review. ***Journal of Manipulative and Physiological Therapeutics***. 29(2): 163-169.
- STANKOVIC, R. & JOHNNELL, O. (1990). Conservative Treatment of Acute Low Back Pain. ***Spine***. 15: 120-123.
- STEVENSON, J.M., WEBER, C.L., SMITH, J.T., DUMAS, G.A. & ALBERT, W.J. (2001). A Longitudinal Study of the Development of LBP in an Industrial Population. ***Spine***. 26: 1370-1377.
- STOKES, I.A., WILDER, D.G., FRYMOYER, J.W. & POPE, M.H. (1981). Volvo Award in Clinical Science. Assessment of Patients with Low Back Pain by Biplanar Radiographic Measurement of Intervertebral Motion. ***Spine***. 6: 233-240.
- SUNDARARAJAN, V., KONRAD, T.R., GARRETT, J. & CAREY, T. (1998). Patterns and Determinants of Multiple Provider Use in Patients with Acute Low Back Pain. ***Journal of General Internal Medicine: Official Journal of The Society for Research and Education in Primary Care Internal Medicine***. 13: 528-533.

- SULLIVAN, M.J.L., BISHOP, S. & PIVIK, J. (1995). The Pain Catastrophising Scale: Development and Validation. *Psychological Assessment*. 7: 524-532.
- SULLIVAN, M.D. (2004). What are the Goals of Pain Treatment? In: DWORKIN, R.H. & BREITBART, W.S. (eds.) **Psychosocial Aspects of Pain: A Handbook for Health Care Providers**. Seattle, WA: IASP Press: 215-230.
- SUNG, P.S., LAMMERS, A.R. & DANIAL, P. (2008). Different Parts of Erector Spinae Muscle Fatigability in Subjects with and without Low Back Pain. *The Spine Journal*. Doi:10.1016/j.spinee.2007.11.001
- SUNI, J.H., OJA, P., MIILUNPALO, S.I., PASANEN, M.E., VUORI, I.M. & BOS, K. (1998). Health-Related Fitness Test Battery for Adults: Association with Perceived Health, Mobility and Back Function and Symptoms. *Archives of Physical Medicine and Rehabilitation*. 79: 559-569.
- SVEBAK, S. (2000). Musculoskeletal Problems and Stress. In: FINK, G. ed. **Encyclopedia of Stress**. Vol II. San Diego, CA: Academic Press.
- SWINKELS, A. & DOLAN, P. (1998). Regional Assessment of Joint Position Sense in the Spine. *Spine*. 23: 590-597.
- SWINKELS-MEEWISSE, I.E., SWINKELS, R.A., VERBEEK, A.L., VLAEYEN, J.W.S. & OOSTENDORP, R.A.B. (2003). Psychometric Properties of the Tampa Scale for Kinesiophobia and the Fear-Avoidance Beliefs Questionnaire in Acute Low Back Pain. *Manual Therapy*. 8:29-36.
- TABACHNICK, B.G. & FIDELL, L.S. (1996). **Using Multivariate Statistics**. (3<sup>rd</sup> Edition). Northridge: HarperCollinsCollege Publishers.



- TAIMELA, S. & HÄRKÄPÄÄ, K. (1996). Strength, Mobility, their Changes, and Pain Reduction in Active Functional Restoration for Chronic Low Back Disorders. ***Journal of Spinal Disorders.*** 9: 306-312.
- TAIMELA, S., KANKAANPÄÄ, M. & LUOTO, S. (1999). The Effect of Lumbar Fatigue on the Ability to Ensure a Change in Lumbar Position: A Controlled Study in Chronic Low Back Pain Patients and Healthy Controls. ***Spine.*** 24: 1322-1327.
- TAIMELA, S, DIEDERICH, C., HUBSCH, M. & HEINICY, M. (2000). The Role of Physical Exercise and Inactivity in Pain Recurrence and Absenteeism from Work after Active Outpatient Rehabilitation for Recurrent or Chronic LBP. ***Spine.*** 25: 1809-1816.
- TAKAYANAGI, K., TAKAHASHI, K., YAMAGATA, M., MORIYA, H., KITAHARA, H. & TAMAKI, T. (2001). Using Cineradiography for Continuous Dynamic-Motion Analysis of the Lumbar Spine. ***Spine.*** 26: 1858-1865.
- TAKEMASA, R., YAMAMOTO, H. & TANI, T. (1995). Trunk Muscle Strength In and Effect of Trunk Muscle Exercises for Patients with Chronic Low Back Pain: The Difference in Patients with and without Organic Lumbar Lesions. ***Spine.*** 20: 2522-2530.
- TALLEY, N.J.& SPILLER, R. (2002). Irritable Bowel Syndrome: A Little Understood Organic Bowel Disease? ***Lancet.*** 360: 555-564.
- TAWFIK, B. (2001). Symmetry and Linearity of Trunk Function in Subjects with Non-Specific Low Back Pain. ***Clinical Biomechanics (Bristol, Avon).***16: 114-120.
- TESH, K.M., SHAWDUNN, J. & EVANS, J.H. (1987). The Abdominal Muscles and Vertebral Stability. ***Spine.*** 12: 501-508.

- TEYHEN, D.S., MILTENBERGER, C.E., DEITERS, H.M., DEL TORO, Y.M., PULLIAM, J.N., CHILDS, J.D., BOYLES, R.E. & FLYNN, T.W. (2005). The Use of Ultrasound Imaging of the Abdominal Drawing-In Maneuver in Subjects with Low Back Pain. ***The Journal of Orthopaedic and Sports Physical Therapy***. 35: 346-355.
- THELANDER, U., FAGERLUND, M., FRIBERG, S. & LARSSON, S. (1992). Straight Leg Raising versus Radiologic Size, Shape, and Position of Lumbar Disc Hernias. ***Spine***. 17(4):395-399.
- THOMAS, E., SILMAN, A.J., CROFT, P.R., PAPAGEORGIOU, A.C., JAYSON, M.I. & MACFARLANE, G.J. (1999). Predict who Develops Low Back Pain in Primary Care: A Prospective Study. ***BMJ***. 318(7199): 1662-1667.
- THOMAS, J.S & FRANCE, C.R. (2007). Pain-Related Fear Is Associated with Avoidance of Spinal Motion During Recovery from Low Back Pain. ***Spine***. 32(16): E460-E466.
- THOMAS, J.R. & NELSON, J.K. (2001). **Research Methods in Physical Activity**. Champaign, IL: Human Kinetics.
- THOMPSON, J.M.T. & HUNT, G.W. (1984). **The General Conservative Theory**. In: Elastic Instability Phenomena. New York, NY: John Wiley & Sons.
- THORSTENSSON, A. & ARVIDSON, A. (1982). Trunk Muscle Strength and Low Back Pain. ***Scandinavian Journal of Rehabilitation Medicine***. 14: 69-75.
- THORSTENSSON, A. & CARLSON, H. (1987). Fibre Types in Human Lumbar Back Muscles. ***Acta Physiologica Scandinavica***. 131: 195-202.

- TODOROV, E. & JORDAN, M.I. (2002). Optimal Feedback Control as a Theory of Motor Coordination. *Nature Neuroscience*. 5: 1226-1235.
- TREEDE, R.D KENSHALO, D.R. GRACELY, R.H. & JONES, A.K.P. (1999). The Cortical Representation of Pain. *Pain*. 79: 105-111.
- TREEDE, R.D., JENSEN, T.S., CAMPBELL, J.N., CRUCCU, G., DOSTROVSKY, J.O., GRIFFIN, J.W., HANSSON, P., HUGHES, R., NURMIKKO, T. & SERRA, J. (2008). Neuropathic Pain: Redefinition and a Grading System for Clinical and Research Purposes. *Neurology*. 70(18): 1630-1635.
- TSAO, H. & HODGES, P.W. (2008). Persistence of Improvements in Postural Strategies Following Motor Control Training in People with Recurrent Low Back Pain. *Journal of Electromyography and Kinesiology*. 18: 559-567.
- TUCCI, J.T., CARPENTER, D.M., POLLOCK, M.I., GRAVES, J.E. & LEGGETT, S.H. (1992). Effect of Reduced Frequency of Training and Detraining on Lumbar Extension Strength. *Spine*. 17(12): 1497-1501.
- TULDER, M.W., VAN ESMAIL, R., BOMBARDIER, C. & KOES, B.W. (2001). Back Schools for Non-Specific Low Back Pain (Cochrane Review). *The Cochrane Library*. 4.
- TURK, D.C. & MARCUS, D.A. (1994). Assessment of Chronic Pain Patients. *Seminars in Neurology*. 14: 206-212.
- TURK, D.C. (2002). Remember the Distinction between Malignant and Benign Pain? Well, Forget It. *Clinical Journal of Pain*. 18: 75-76.

TURNER, J.A. (1996). Educational and Behavioral Interventions for Back Pain in Primary Care. *Spine*. 21: 2851-2859.

TWOMEY, L.T. & TAYLOR, J.R. (1987). Age Changes in Lumbar Vertebrae and Intervertebral Discs. *Clinical Orthopaedics and Related Research*. 224: 97-104.

VALENCIA, F.P. & MUNRO, R.R. (1985). An Electromyographic Study of the Lumbar Multifidus in Man. *Electromyography and Clinical Neurophysiology*. 25: 205-221.

VAN DEN HOOGEN, H., KOES, B.W., DEVILLE, W., VAN EIJK, J.T. & BOUTER, L.M. (1996). The Inter-Observer Reproducibility of Lasegue's Sign in Patients with Low Back Pain in General Practice. *The British Journal of General Practice: The Journal of the Royal College of General Practitioners*. 46(413): 727-730.

VAN DEN HOOGEN, H.J., KOES, B.W., DEVILLE, W., VAN EIJK, J.T. & BOUTER, L.M. (1997). The Prognosis of Low Back Pain in General Practice. *Spine*. 22(13): 1515-1521.

VAN DER VELDE, G. & MIERAU, D. (2000). The Effect of Exercise on Percentile Rank Aerobic Capacity, Pain, and Self-Rated Disability in Patients with Chronic Low Back Pain: A Retrospective Chart Review. *Archives of Physical Medicine and Rehabilitation*. 81: 1457-1463.

VAN DIEEN, J.H. & HEIJBLON, P. (1996). Reproducibility of Isometric Trunk Extension Torque, Trunk Extensor Endurance, and Related Electromyographic Parameters in

the Context of their Clinical Applicability. ***Journal of Orthopaedic Research: Official Publication of the Orthopaedic Research Society.*** 14: 139-143.

VAN TULDER, M.W., KOES, B.W. & BOUTER, L.M. (1997). Conservative Treatment of Acute and Chronic Nonspecific Low Back Pain: A Systematic Review of Randomized Controlled Trails of the Most Common Interventions. ***Spine.*** 22:2128-2156.

VAN TULDER, M., MALMIVAARA, A., ESMAIL, R. & KOES, B. (2000). Exercise Therapy for Low Back Pain: A Systematic Review within the Framework of the Cochrane Collaboration Back Review Group. ***Spine.*** 25: 2784-2796.

VAN TULDER, M.W., OSTELO, R., VLAEYEN, J.W., LINTON, S.J., ORLEY, S.J. & ASSENDELFT, W.J. (2001). Behavioral Treatment for Chronic LBP: A Systematic Review within the Framework of the Cochrane Back Review Group. ***Spine.*** 26: 270-281.

VAN TULDER, M.W., MALMIVAARA, A., ESMAIL, R. & KOES, B.W. (2004). **Exercise Therapy for Low Back Pain (Cochrane Review). The Cochrane Library, Issue 3.** Chichester, UK: John Wiley & Sons Ltd.

VAN VLIET, P. & HENEGHAN, N. (2006). Motor Control and the Management of Musculoskeletal Dysfunction. ***Manual Therapy.*** 3: 208-213.

VAN VUUREN, B.J., BECKER, P., VAN HEERDEN, H.J., ZINZEN, E. & MEEUSEN, R. (2005). Lower Back Problems and Occupational Risk Factors in a South African Steel Industry. ***American Journal of Industrial Medicine.*** 47: 451-457.

- VERBUNT, J.A., SEELEN, H.A., VLAEYEN, J.W., VAN DE HEIJDEN, G.J., HEUTS, P.H., PONS, K. & KNOTTNERUS, J.A. (2003). Disuse and Deconditioning in Chronic Low Back Pain: Concepts and Hypotheses on Contributing Mechanisms. *European Journal of Pain*. 7: 9-21.
- VERDU, B., DECOSTERD, I., BUCLIN, T, STIEFEL, F. & BERNEY, A. (2008). Antidepressants for the Treatment of Chronic Pain. *Drugs*. 68(18): 2611-2632.
- VIDEMAN, T., SAMA, S., BATTIE, M.C., KOSKINEN, S., GILL, K., PAANANEN, H. & GIBBONS, L. (1995). The Long Term Effects of Physical Loading and Exercise Lifestyles on Back-Related Symptoms, Disability, and Spinal Pathology among Men. *Spine*. 20: 699-709.
- VOLINN, E. (1997). The Epidemiology of Low Back Pain in the Rest of the World. A Review of Surveys in Low- and Middle-Income Countries. *Spine*. 22: 1747-1754.
- VON KORFF, M. & SAUNDERS, K. (1996). The Course of Back Pain in Primary Care. *Spine*. 21: 2833-2837.
- VON KORFF, M., CRANE, P., LANE, M., MIGLIORETTI, D.L., SIMON, G., SAUNDERS, K., STANG, P., BRANDENBURG, N. & KESSLER, R. (2005). Chronic Spinal Pain and Physical-Mental Comorbidity in the United States: Results from the National Comorbidity Survey Replication. *Pain*. 113: 331-339.
- VLAEYEN, J.W., HAAZEN, I.W., SCHUERMAN, J.A. (1995). Behavioral Rehabilitation of Chronic Low Back Pain: Comparison of an Operant Treatment, an Operant-Cognitive Treatment and an Operant-Respondent Treatment. *The British Journal of Clinical Psychology*. 34: 95-118.

VLAEYEN, J.W., KOLE-SNIJDERS, A.M., BOEREN, R.G. & VAN EEK, T. (1995). Fear of Movement / (re)injury in Chronic Low Back Pain and its Relation to Behavioral Performance. *Pain*. 62(3): 363-372.

VLAEYEN, J.W. & LINTON, S.J. (2000). Fear-Avoidance and its Consequences in Chronic Musculoskeletal Pain: A State of the Art. *Pain*. 85: 317-332.

VLAEYEN, J.W.S. & MORLEY, S. (2005). Cognitive-Behavioral Treatments for Chronic Pain. What Works for Whom? *The Clinical Journal of Pain*. 21(1): 1-8.

WADDELL, G.; NEWTON, M., HENDERSON, I., SOMERVILLE, D. & MAIN, C.J. (1993). A Fear-Avoidance Beliefs Questionnaire (FABQ) and the Role of Fear-Avoidance Beliefs in Chronic Low Back Pain and Disability. *Pain*. 52: 157-168.

WADDELL, G. (1996). Low Back Pain: A Twentieth Century Health Care Enigma. *Spine*. 21: 2820-2825.

WADDELL, G., FEDER, G., MCINTOSH, A., HUTCHINSON, A. & LEWIS, M. (1996). **Low Back Pain Evidence Review**. London, UK: RoyalCollege of General Practitioners.

WADDELL, G., FEDER, G., LEWIS, M. (1997). Systematic Reviews of Bed Rest and Advice to Stay Active for Acute Low Back Pain. *The British Journal of General Practice: The Journal of the RoyalCollege of General Practitioners*. 47: 647-652.

WADDELL, G. (2004). **The Back Pain Revolution**. Edinburgh, UK: Churchill-Livingstone.

WALKER, B. (2000). The Prevalence of Low Back Pain: A Systematic Review of the Literature from 1966 to 1998. *Journal of Spinal Disorders*. 13(3): 205-217.

WALKER, B., MULLER, R. & GRANT, W. (2006). Low Back Pain in Australian Adults. Prevalence and Associated Disability. ***Journal of Manipulative and Physiological Therapeutics***. 27: 238-244.

WALL, P. & MELZACK, R. (1999). **Textbook of Pain**. (4<sup>th</sup> Edition). London, UK: Churchill-Livingstone.

WALLBOM, A.S, GEISSER, M.E., HALG, A.J., YAMAKAWA, K. & MONTGOMERY, D. (2002). Concordance between Rating of Perceived Exertion and Function in Persons with Chronic, Disabling Back Pain. ***Journal of Occupational Rehabilitation***. 12(2): 93-98.

WALSH, J., FLATLEY, M., JOHNSTON, N. & BENNETT, K. (2007). Slump Test: Sensory Response in Asymptomatic Subjects. ***The Journal of Manual & Manipulative Therapy***. 15(4): 231-238.

WEWERS, D.A. & LOWE, N.K. (1990). A Critical Review of Visual Analogue Scales in the Measurement of Clinical Phenomena. ***Research in Nursing & Health***. 13: 227-236.

WIKIPEDIA, [http://en.wikipedia.org/wiki/Mann-Whitney\\_U](http://en.wikipedia.org/wiki/Mann-Whitney_U)

WIKIPEDIA, [http://en.wikipedia.org/wiki/Wilcoxon\\_signed-rank\\_test](http://en.wikipedia.org/wiki/Wilcoxon_signed-rank_test)

WILDER, D.G., ALEKSIEV, A.R., MAGNUSSON, M.L., POPE, M., SPRAT, K. & GOEL, V. (1996). Muscular Response to Sudden Load: A Tool to Evaluate Fatigue and Rehabilitation. ***Spine***. 21: 2628-2639.



- WILKE, H.J., WOLF, S., CLAES, L.E., ARAND, M. & WIESEND, A.(1995). Stability Increase of the Lumbar Spine with Different Muscle Groups: A Biomechanical *In Vitro* Study. ***Spine***. 20: 192-198.
- WILLIAMS, J.G. & ESTON, R.G. (1996). Exercise Intensity Regulation. In ESTON, R. & REILLY, T. (1996). **Kinanthropometry and Exercise Physiology Laboratory Manual**. London, UK:E & FN SPON.
- WILLIAMS, K., STEINBERG, L. & PETRONIS, J. (2003). Therapeutic Application of Iyengar Yoga for Healing Chronic Low Back Pain. ***International Journal of Yoga Therapy***. 13: 55-67.
- WILLIAMS, P.L., WARWICK, R., DYSON, M. & BANNISTER, L.H. (Eds.) (1989). **Gray's Anatomy**. (37<sup>th</sup> edition). London, UK: Churchill Livingstone.
- WOLF, S.L., WOLF, L.B. & SEGAL, R.L. (1989). The Relationship of Extraneous Movements to Lumbar Paraspinal Muscle Activity: Implications for EMG Biofeedback Training Applications to Low Back Pain Patients. ***Biofeedback and Self-Regulation***. 14: 63-74.
- WOOLF, A. & PFLEGER, B. (2003). Burden of Major Musculoskeletal Conditions. ***Bulletin of the World Health Organization***. 81(9): 646-656.
- WOOLF, C.J. (1983). Evidence for a Central Component of Post-Injury Pain Hypersensitivity. ***Nature***. 306: 686-688.
- WOOLF, C.J., SHORTLAND, P. & COGGESHALL, R.E. (1992). Peripheral Nerve Injury Triggers Central Sprouting of Myelinated Afferents. ***Nature***. 355: 75-78.

- WOOLF, C.J. (2004). Pain: Moving from Symptom Control towards Mechanism – Specific Pharmacological Management. *Annual International Journal of Medicine*. 140: 441-451.
- WORKU, Z. (2000). Prevalence of Low Back Pain in Lesotho Mothers. *Journal of Manipulative and Physiological Therapeutics*. 23(3): 147-154.
- WRIGHT, A., LLOYD-DAVIES, A., WILLIAMS, ELLIS, R. & STRIKE, P. (2005). Individual Active Treatment Combined with Group Exercise for Acute and Subacute Low Back Pain. *Spine*. 30(11): 1235-1241.
- YOSHIHARA, K., SHIRAI, Y., NAKAYAMA, Y. & UESAKA, S. (2001). Histochemical Changes in the Multifidus Muscle in Patients with Lumbar Intervertebral Disc Herniation. *Spine*. 26(6): 622-626.
- YU, J., WINLOVE, C.P., ROBERTS, S. & URBAN, J.P. (2002). Elastic Fibre Organization in the Intervertebral Discs of the Bovine Tail. *Journal of Anatomy*. 201: 465-475.
- ZACHRISSON-FORSSELL, M. (1981). The BackSchool. *Spine*. 6: 104-106.
- ZATTARA, M. & BOUISSET, S. (1988). Posturo-Kinetic Organisation during the Early Phase of Voluntary Upper Limb Movement: I. Normal Subjects. *Journal of Neurology, Neurosurgery, and Psychiatry*. 51: 956-965.

## **Annexure A**

### **Questionnaires**

1. VAS Pain Score
2. Tampa Scale
3. Oswestry Disability Index
4. Functional Rating Index
5. Fear Avoidance Beliefs Questionnaires
6. Exercise Intensity Progression Measurement
7. DN4 Questionnaire

## Visual Analog Scale (VAS)

Please indicate the amount of pain recently experienced by marking an (X) through the line.

100mm VAS scale – Left hand marker “no pain”, right hand marker “extreme pain”.

|—————|

No pain Extreme Pain



Tampa Scale for Kinesiophobia  
(Miller, Kori and Todd 1991)

1 = strongly disagree  
2 = disagree  
3 = agree  
4 = strongly agree

1. I'm afraid that I might injury myself if I exercise	1	2	3	4
2. If I were to try to overcome it, my pain would increase	1	2	3	4
3. My body is telling me I have something dangerously wrong	1	2	3	4
4. My pain would probably be relieved if I were to exercise	1	2	3	4
5. People aren't taking my medical condition seriously enough	1	2	3	4
6. My accident has put my body at risk for the rest of my life	1	2	3	4
7. Pain always means I have injured my body	1	2	3	4
8. Just because something aggravates my pain does not mean it is dangerous	1	2	3	4
9. I am afraid that I might injure myself accidentally	1	2	3	4
10. Simply being careful that I do not make any unnecessary movements is the safest thing I can do to prevent my pain from worsening	1	2	3	4
11. I wouldn't have this much pain if there weren't something potentially dangerous going on in my body	1	2	3	4
12. Although my condition is painful, I would be better off if I were physically active	1	2	3	4
13. Pain lets me know when to stop exercising so that I don't injure myself	1	2	3	4
14. It's really not safe for a person with a condition like mine to be physically active	1	2	3	4
15. I can't do all the things normal people do because it's too easy for me to get injured	1	2	3	4
16. Even though something is causing me a lot of pain, I don't think it's actually dangerous	1	2	3	4
17. No one should have to exercise when he/she is in pain	1	2	3	4

Reprinted from:  
*Pain*, Fear of movement/(re) injury in chronic low back pain and its relation to behavioral performance, 62, Vlaeyen, J., Kole-Snijders A., Boeren R., van Eek H., 371.  
Copyright (1995) with permission from International Association for the Study of Pain.



## Oswestry Disability Questionnaire

This questionnaire has been designed to give us information as to how your back or leg pain is affecting your ability to manage in everyday life. Please answer by checking **one box in each section** for the statement which best applies to you. We realise you may consider that two or more statements in any one section apply but please just shade out the spot that indicates the statement **which most clearly describes your problem**.

### Section 1: Pain Intensity

- I have no pain at the moment
- The pain is very mild at the moment
- The pain is moderate at the moment
- The pain is fairly severe at the moment
- The pain is very severe at the moment
- The pain is the worst imaginable at the moment

### Section 2: Personal Care (eg. washing, dressing)

- I can look after myself normally without causing extra pain
- I can look after myself normally but it causes extra pain
- It is painful to look after myself and I am slow and careful
- I need some help but can manage most of my personal care
- I need help every day in most aspects of self-care
- I do not get dressed, wash with difficulty and stay in bed

### Section 3: Lifting

- I can lift heavy weights without extra pain
- I can lift heavy weights but it gives me extra pain
- Pain prevents me lifting heavy weights off the floor but I can manage if they are conveniently placed eg. on a table
- Pain prevents me lifting heavy weights but I can manage light to medium weights if they are conveniently positioned
- I can only lift very light weights
- I cannot lift or carry anything

### Section 4: Walking\*

- Pain does not prevent me walking any distance
- Pain prevents me from walking more than 2 kilometres
- Pain prevents me from walking more than 1 kilometre
- Pain prevents me from walking more than 500 metres
- I can only walk using a stick or crutches
- I am in bed most of the time

### Section 5: Sitting

- I can sit in any chair as long as I like
- I can only sit in my favourite chair as long as I like
- Pain prevents me sitting more than one hour
- Pain prevents me from sitting more than 30 minutes
- Pain prevents me from sitting more than 10 minutes
- Pain prevents me from sitting at all

### Section 6: Standing

- I can stand as long as I want without extra pain
- I can stand as long as I want but it gives me extra pain
- Pain prevents me from standing for more than 1 hour
- Pain prevents me from standing for more than 30 minutes
- Pain prevents me from standing for more than 10 minutes
- Pain prevents me from standing at all

### Section 7: Sleeping

- My sleep is never disturbed by pain
- My sleep is occasionally disturbed by pain
- Because of pain I have less than 6 hours sleep
- Because of pain I have less than 4 hours sleep
- Because of pain I have less than 2 hours sleep
- Pain prevents me from sleeping at all

### Section 8: Sex Life (if applicable)

- My sex life is normal and causes no extra pain
- My sex life is normal but causes some extra pain
- My sex life is nearly normal but is very painful
- My sex life is severely restricted by pain
- My sex life is nearly absent because of pain
- Pain prevents any sex life at all

### Section 9: Social Life

- My social life is normal and gives me no extra pain
- My social life is normal but increases the degree of pain
- Pain has no significant effect on my social life apart from limiting my more energetic interests e.g. sport
- Pain has restricted my social life and I do not go out as often
- Pain has restricted my social life to my home
- I have no social life because of pain

### Section 10: Travelling

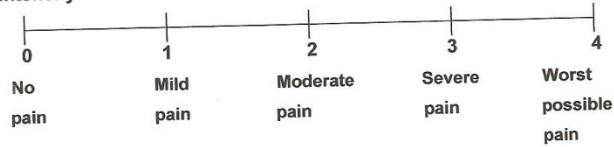
- I can travel anywhere without pain
- I can travel anywhere but it gives me extra pain
- Pain is bad but I manage journeys over two hours
- Pain restricts me to journeys of less than one hour
- Pain restricts me to short necessary journeys under 30 minutes
- Pain prevents me from travelling except to receive treatment



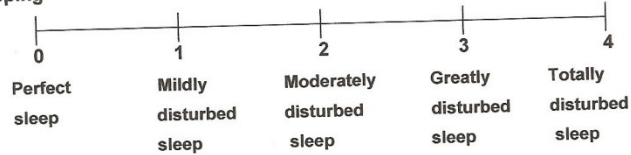
### Functional Rating Index

In order to properly assess your personal condition, we must understand how much your back problems have affected your ability to manage everyday activities. For each item below, please cross the number which most closely describes your condition now.

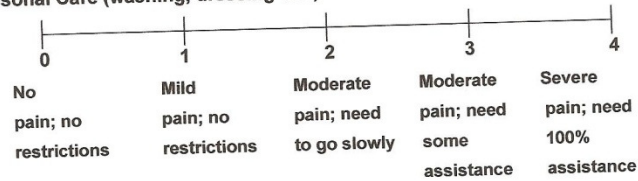
#### 1. Pain Intensity



#### 2. Sleeping

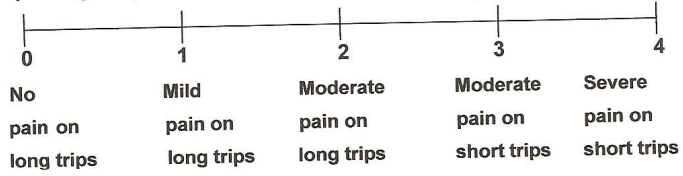


#### 3. Personal Care (washing, dressing etc.)

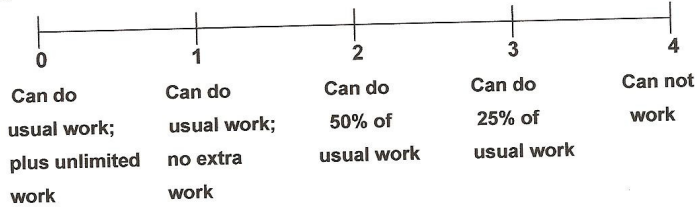




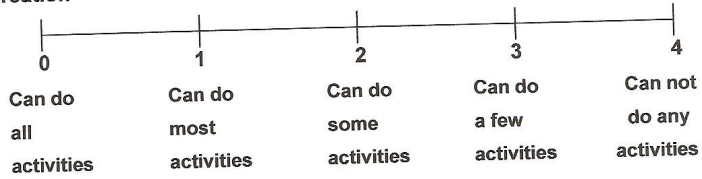
**4. Travel (driving etc.)**



**5. Work**



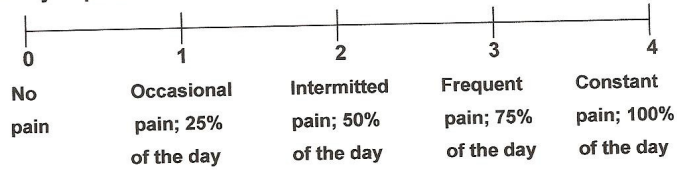
**6. Recreation**



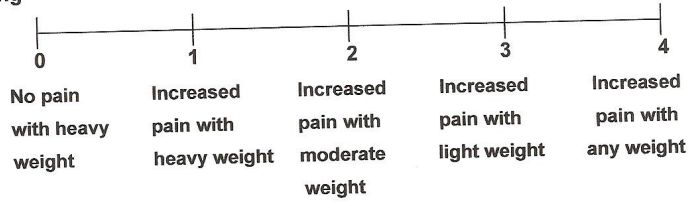




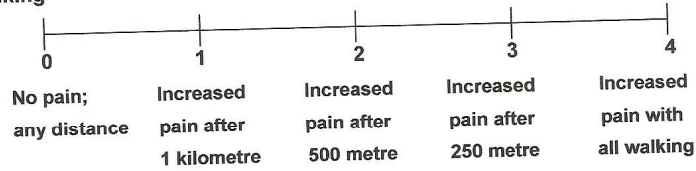
7. Frequency of pain



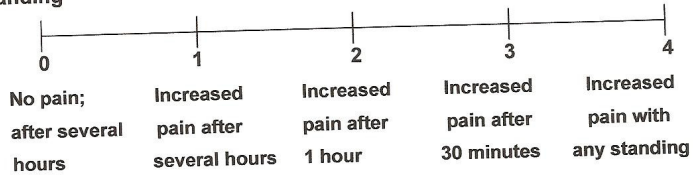
8. Lifting



9. Walking



10. Standing



**FABQ**

Here are some of the things which other people have told us about their pain. For each statement please cross the number to say how much physical activities such as bending, lifting, walking or driving affect or would affect YOUR back trouble.

0            1            2            3            4            5            6

**Completely  
disagree (CD)**

**Unsure (U)**

**Completely  
agree (CA)**

		<b>CD</b>			<b>U</b>			
<b>CA</b>								
1.	My pain was caused by physical activity.	0	1	2	3	4	5	6
2.	Physical activity makes my pain worse.	0	1	2	3	4	5	6
3.	Physical activity may harm my back.	0	1	2	3	4	5	6
4.	I should not do physical activities which (might) make my pain worse.	0	1	2	3	4	5	6
5.	I cannot do physical activities which (might) make my pain worse.	0	1	2	3	4	5	6

**The following statements are about how your NORMAL work affect your back pain.**

		<b>CD</b>			<b>U</b>				<b>CA</b>				
6.	My pain was caused by my work or by an accident at work.	0	1	2	3	4	5	6					
7.	My work aggravated my trouble.	0	1	2	3	4	5	6					
8.	I have claim for compensation for my pain.	0	1	2	3	4	5	6					



**CA** **CD** **U**

9.	My work is too heavy for me.	0	1	2	3	4	5	6
10.	My work makes or would make my pain worse.	0	1	2	3	4	5	6
11.	My work might harm my back.	0	1	2	3	4	5	6
12.	I <u>should</u> not do my normal work with my present pain.	0	1	2	3	4	5	6
13.	I <u>cannot</u> do my normal work with my present pain.	0	1	2	3	4	5	6
14.	I cannot do my normal work till my pain is treated.	0	1	2	3	4	5	6
15.	I do not think that I will be able to work <u>normally</u> within 3 months.	0	1	2	3	4	5	6
16.	I do not think that I will ever be able to do my present work <u>normally</u> .	0	1	2	3	4	5	6

### Exercise Intensity Progression Measurement:

Please use the Borg RPE scale provided to rate the intensity (difficulty) of the exercises as they progressed from one programme to the next

Exercise	Programme 1	Programme 2	Programme 3
Cycling			
Lat Pulldown			
Cable Exercise			
Side Bridging			
Hip Lifts			
Alt Superman			
Abdominal Crunch			

### Borg RPE Scale

6 No exertion at all	14
7 Extremely light	15 Hard (Heavy)
8	16
9 Very light	17 Very hard
10	18
11 Light	19 Extremely hard
12	20 Maximal exertion
13 Somewhat hard	

Gunnar Borg, 1998, *Borg's Perceived Exertion and Pain Scales, Human Kinetics, Champaign, IL.*



## DN4 – QUESTIONNAIRE

To estimate the probability of neuropathic pain, please answer yes or no for each item of the following four questions.

### INTERVIEW OF THE PATIENT

#### QUESTION 1:

Does the pain have one or more of the following characteristics?	YES	NO
Burning .....	<input type="checkbox"/>	<input type="checkbox"/>
Painful cold .....	<input type="checkbox"/>	<input type="checkbox"/>
Electric shocks .....	<input type="checkbox"/>	<input type="checkbox"/>

#### QUESTION 2:

Is the pain associated with one or more of the following symptoms in the same area?	YES	NO
Tingling .....	<input type="checkbox"/>	<input type="checkbox"/>
Pins and needles .....	<input type="checkbox"/>	<input type="checkbox"/>
Numbness .....	<input type="checkbox"/>	<input type="checkbox"/>
Itching .....	<input type="checkbox"/>	<input type="checkbox"/>

### EXAMINATION OF THE PATIENT

#### QUESTION 3:

Is the pain located in an area where the physical examination may reveal one or more of the following characteristics?	YES	NO
Hypoesthesia to touch .....	<input type="checkbox"/>	<input type="checkbox"/>
Hypoesthesia to pinprick .....	<input type="checkbox"/>	<input type="checkbox"/>

#### QUESTION 4:

In the painful area, can the pain be caused or increased by:	YES	NO
Brushing? .....	<input type="checkbox"/>	<input type="checkbox"/>

YES = 1 point

NO = 0 points

Patient's Score: /10



## **Annexure B**

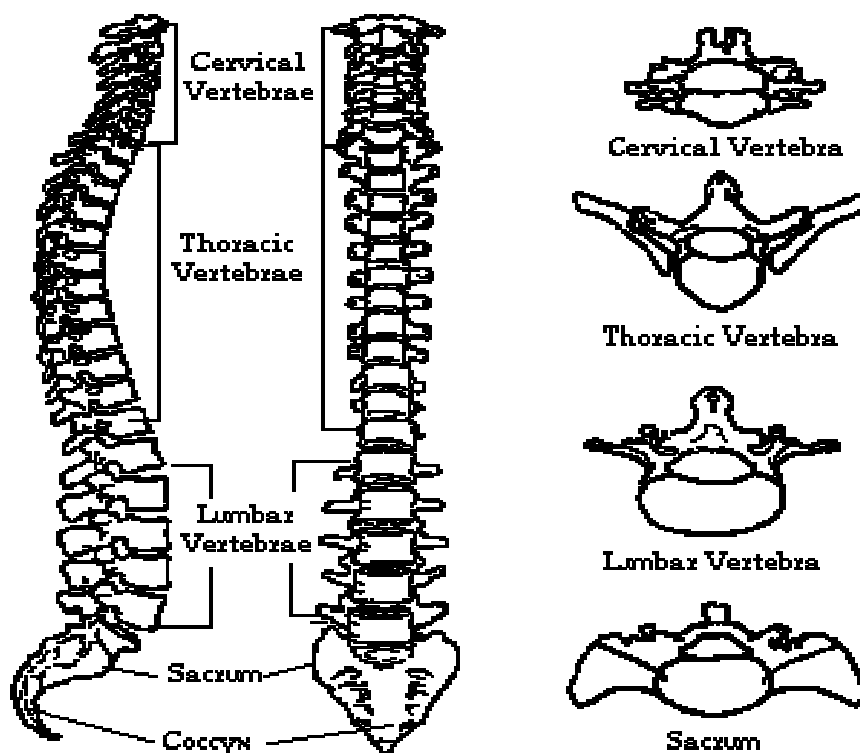
### **BackSchool**

# The Only Information You will Ever Need to Treat Your Back

## Pain

- **Discussion of correct and proper anatomy:** These discussions will focus on the involved anatomical structures of the lower back, and their possible influence in the cause of their problems.

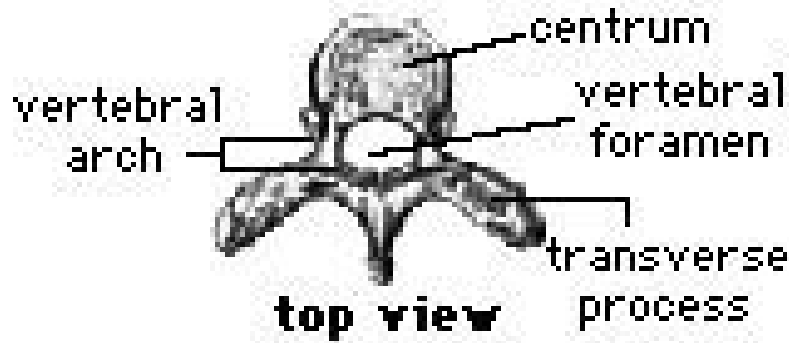
The vertebral column is the starting point. It is composed of many bony parts called **vertebrae**, which are separated from each other by masses of fibro-cartilage called intervertebral disks.



The vertebral column supports the head and trunk of the body, yet is flexible enough to permit movements, such as bending forward and backwards, to the sides and rotation.

A typical vertebra has a drum-shaped body (centrum), which forms the thick anterior portion of the vertebrae.

## Vertebra



The intervertebral disks that separates adjacent vertebra are fastened to the roughened upper and lower surfaces of the vertebral bodies. These disks cushion and soften the forces caused by such movements as walking and jumping, which might otherwise fracture vertebrae or injure the brain. Projecting posteriorly (backwards) from each vertebral body are two stalks called **pedicles**, which forms the sides of the vertebral foramen. Two plates called **laminae** arise from the pedicles and fuse in the back to become a **spinous process**. The pedicles, laminae and spinous process together complete a bony **vertebral arch**, around the vertebral foramen, through which the spinal cord passes.

Important muscles include the extensors (longissimus, iliocostalis, and Multifidus groups), the abdominal muscles (rectus abdominis, external oblique, internal oblique and transverse abdominis) and the Quadratus lumborum. The abdominal muscles work together but also independently. The obliques activate differentially by creating twisting forces and can enhance forward flexion. Rectus abdominis is primarily a flexor. The obliques together with transverse abdominis form a containing belt around the entire abdomen resulting in a stiffening force that assists with spinal stability.

The quadratus lumborum (QL) is by design an effective lateral (sideways) stabilizing muscle. The QL seems to be active during a variety of flexion-dominant Forward-bending), extensor-dominant (backwards bending) and lateral bending (side bending) tasks.

The extensor muscles provide assistance during extension (backwards bending) and seem to be most effective during movements that involve hip bending rather than back bending.



- **The Importance of Stabilization (Bracing):** This will explain the concept of lumbar stabilization and how to achieve it.

Stability is very important for low back health. Tissue damage results because of joint laxity, and this then leads to joint instability. Ligament failure for example, causes joint instability under load and with motion. Injuries such as end-plate fractures with a loss of disc height can also cause tissue damage that will result in unstable joint behavior. Therefore, to summarize, **instability can both be caused by and be the result of injury**. The goal of stability is to activate the target muscles that are responsible for achieving lumbar stability, and to then maintain that stability. This will then help to prevent future injuries by decreasing the incidence of tissue injuries as a result of instability.

Stability is achieved by the simultaneous activation of the transvers abdominis, multifidus, quadratus lumborum and oblique muscles, which forms the so-called ‘core stabilizers.’ These muscles have been scientifically shown to act as a shock absorber during movements, which lessens the stress placed on the lower back. Stability is achieved by visualizing and then contracting the muscles of the pelvic floor, which in turn activates the correct muscles. This contracture is then held during movements to ensure that the stabilizing muscles take the pressure off the lower back. This maneuver is also called abdominal bracing. When performed correctly, there will be no visible changes in the abdominal wall. The necessary muscle needs only to be activated rather than pulling in the whole abdominal wall.

- **Discussion of proper ergonomics:** Certain tasks and movements in everyday life can worsen lower back problems. These discussions will focus on proper lifting and application techniques.
- When bending to pick up an object, always try to keep the back straight and bend the knees.
  - When you have to bend forward and you cannot bend the knees, choose to rather bend at the hips and still keep the back straight.
  - When you have to bend forward, remember, that bending forward with the back fully rounded will increase the risk of back trouble. Always try to bend at the hips as this will decrease the pressure on the spine.
  - Remember, when performing any type of movement, it is very important to always remember to perform abdominal bracing or stabilizing.
  - When picking up a light object off the floor, using the so-called golfers pick-up can be of great benefit as it reduces the pressure on the spine as well as the knees.
  - When pushing objects, such as vacuum cleaners, try to push with the hands through the low back as to effectively direct the forces.
  - Avoid twisting motions when performing activities like vacuuming. Direct the forces through the low back to minimize the load on the spine.
  - When performing an activity like shoveling, resting the hand on the front leg redirects the forces directly to the ground and by-passes the arm and spine linkage.
  - When you have to spend a long time standing, don't immediately pick up a heavy object. Stand up first and walk around for a bit before you pick up the object.
  - Always carry objects close to your body.

➤ **Avoiding bed rest and remaining active with normal activities that were avoided**

**because of back pain:** Bed rest is detrimental to lower back problems, and these discussions will focus on the importance of avoiding bed rest.

For years, bed rest has been prescribed for patients. However, in recent years, this practice has been questioned by science, due to the lack of evidence to support the use of bed rest. Bed rest has become some kind of medical dogma, where the use is based solely on habit rather than fact. Medical practitioners even prescribe bed rest for patients who don't think that it is necessary. However, regardless of this, bed rest seems to be a favourite treatment tool for lower back pain. However, The International Paris Task Force on Low Back Pain clearly states that bed rest is contraindicated for chronic low back pain. Bed rest should only be prescribed in the most severe cases, and only for a short period of time. Even then, it should be for no longer than 3 days, after which patients should be strongly encouraged to resume their normal daily activities. This is in part due to the fact that degenerative changes start to set in almost immediately, with the spinal stabilizers suffering from weakening and atrophy (wasting). But by staying active and continuing with activities of daily living as tolerated, this wasting is minimized.

It is because of this that bed rest has to as little as possible when back pain is present. Daily activities have to be continued as tolerated and a person has to go on with their lives.

Research has also shown that that bed rest reduces the applied load below the disc osmotic pressure, resulting in a net inflow of fluid. It has been shown that growth in spine length over the usual 8 hours of sleep and then continued bed rest for another period of 32 hours or more is sustained pressure and is suspected to cause back pain. It is proposed that the spine is then stimulated to lay down new bone in response to the higher loads, in this case the higher loads

are due to the swollen discs. This then is interpreted as lying in bed for periods longer than 8 hours actually puts large amounts of stress onto the spine.

- **Discussion of LBP history:** Previous low back pain injury is a major cause factor for future events. The importance of avoiding risk factors for this reason will be discussed.

Past episodes of low back pain is one of the most telling factors for future episodes of low back pain. It is thus very important to take into account previous back pain episodes in order to assess the future likelihood of back pain episodes. This will give a clear picture of factors to take into account.

- 1) The type of injury has to be taken into account.
- 2) The length of the back pain episode

- **Discussion of risk factor prevention:** Risk factor prevention will drastically decrease the chances of suffering from a future back pain event. Different prevention factors will be discussed here.

It is important to understand that there certain risk factors that increases the likelihood of suffering from low back pain. These risk factors could be anything from incorrect posture to the tasks of everyday living. It is important to identify these factors to then be able to properly modify them or avoiding them as necessary. The following risk factors have been identified:

1. Static work posture, specifically prolonged trunk flexion and a twisted or side-ways bent trunk posture.
2. **Seated working posture:** Prolonged sitting is problematic for the low back, and this situation is not fully understood in the occupational world. Research has shown that there is an increased incidence of low back injuries with sedentary occupations. The general view is that sitting is easier on the back, but this is not the case. It has been shown with research that there is an increase in pressure on the disks with sitting posture as well as an increase in strain on the annulus. The spinal stabilizing muscles also tend to relax, and thus increase the pressure on the disks because the muscles then handle a much smaller load than which they are supposed to.

Many occupations require prolong periods of sitting, so sitting is unavoidable. A set of guidelines has been developed to decrease the problems caused by prolonged sitting:

- It is better to have more than one sitting posture, as there is no single best sitting posture and then change to another posture every 10 minutes. The idea behind this is to spread the

pressure of sitting to all structures, in order to have all of the structures carry a little load than to have only the same structures carry all of the load.

- Get out of the chair! Sometimes the best advice is to simply get up and take a break every 30-45 minutes.
- Perform some form of exercise during the course of the day.

3. Frequent torso motion, higher spine rotational velocity and spinal rotation deviations.

4. **Frequent lifting, pushing and pulling.**

5. **Vibration exposure, particularly seated whole-body vibration:** Vibration is linked to elevated rates of low back pain, as there is a loss of stabilization capability with exposure to vibration forces.

6. Peak and cumulative low back shear force, compressive force, and extensor moment.

7. **Incidence of slips and falls:** Falling, especially on the behind can increase the risk for prolonged disability, as this position can rupture ligaments in the lower back and hip.

8. **Repeated full lumbar flexion:** This type of motion is to be avoided, as repeated full lumbar flexion with only moderate loads has been associated with disc herniation ('slipped disc'). Spondylitic fractures can also be caused by repeated stress-strain reversals associated with full flexion.

9. **Time of day (or time after getting up from bed):** You should not engage in any type full bending activities when rising from bed, as the spine has spent the night in a position of full flexion and flexing even more would elevate the resulting stresses dangerously high.

10. Excessive magnitude and repetition of compressive loads, shear loads and torsional displacement and moments.
11. Insufficient loading so that tissue strength is compromised.
12. Lack of lower torso muscle endurance.
13. Perturbed motor control patterns.
14. Age.
15. Gender.
16. Abdominal girth

- **Importance and benefits of exercise:** Exercise therapy is regarded as one of the mainstay treatments for chronic low back pain. These discussions will highlight the importance and benefits of exercise therapy, as well as the safety of different exercises.

It is recommended in the scientific literature that patient suffering from chronic low back pain should take part in a structured, therapeutic exercises programme, regardless of the type of exercise done. Exercise is thus strongly recommended above most common therapies. It is also recommended that the exercise programme combine strength training, stretching and fitness training. Research has shown that a well-chosen exercise programme is a powerful tool for preventing occupational low back problems, as this will help to create a stable spine maintained with healthy and wise motor patterns, along with high levels of muscular endurance is protective for the low back.

- **Work to achieve an internal locus of control:** Internal locus of control is correlated with a quicker and more complete recovery. These discussions will be used to try and facilitate this change of perception in the subjects.

Research has shown that thoughts and beliefs may alter behavior by their direct influence on emotional and physical responses, and individuals may thus become active participants in their treatment if they learn skills to deal with their problems. People are perceived to have either an internal or external locus of control. An internal locus of control refers to a person's feeling of control over their own lives. In contrast, an external locus of control is characterized by a perception that a person's life is controlled by factors beyond their control, for example fate, luck and the influence of other people.



- **Pain Coping Strategies:** This section will deal with effective strategies for pain management.

During treatment it has been shown by research that patients who consider that the treatment that they are offered is highly creditable in helping them cope with their pain problem will do much better than people who are not so convinced by the helpfulness of their treatment modalities.

Research has shown that people who tend to perceive pain in a threatening, catastrophic manner as in the assumption of tissue damage, are more likely to experience pain-related fear and anxiety, and will consequently engage in escape or avoidance behavior. Over time, avoiding of activities of everyday life that are perceived to increase pain and tissue damage is repeatedly reinforced by avoiding activities, and this then contributes to symptoms of disuse and disability. This however, is not always the case. Proper patient education and the interest of the patient towards their back pain and the need to understand the truth behind their pain will be an invaluable tool in understanding that activities of daily living has to be continued with proper technique application.

There needs to be a cultivation of greater objectivity, so that cognitive/emotional alarm reactions to painful situations (e.g. “I’ll never survive this.....” or “This pain will probably go on forever.....”) will become less all-consuming or overwhelming. The process of evaluating these tendencies of the mind to judge whether it is attractive or aversive sensory experience may result in a deconditioning of the alarm reactivity to primary sensations such as physical pain. Thus, while the physical experience of pain may remain largely unchanged, the emotional and cognitive components of the pain experience may be significantly diminished, resulting in less suffering and distress.

Research suggests that the psychological construct of control (e.g. sense of control, self-efficacy) may have important implications for mental and physical health including the management of pain. Research also suggests that actual as well perceived control of pain lessens its impact.

## **Annexure C**

### **Informed Consent**



Pretoria 0002 Republic of South Africa  
<http://www.up.ac.za>

## **FACULTY OF HUMANITIES**

Dept Biokinetics, Sport and Leisure Sciences  
Tel: 012- 420-6040 Fax: 012-420-6099  
[www.bsl.up.ac.za](http://www.bsl.up.ac.za)

## **INFORMED CONSENT**

I \_\_\_\_\_  
(Full name of prospective participant)

have been informed of the procedures and requirements to participate in a research project with title **“Effect of an Aggressive versus Conservative, Multi-Modal Rehabilitation Programme on Non-Specific Chronic Lower Back Pain”**., to be conducted at the University of Pretoria.

### **NATURE AND PURPOSE OF THE STUDY**

You are being asked to participate in a scientific research project. The aim of this study is to learn more about the effects of progressive strength training on chronic non-specific low back pain. Research has shown exercise therapy to be effective, and this study will be used to determine how to better use exercise therapy in the treatment of pain and disability symptoms. Traditional exercise programmes have been found to be effective in the treatment of chronic non-specific low back pain. With this research project, we are hoping to make the exercise programmes more effective.

### **EXPLANATION OF PROCEDURES TO FOLLOW**

Your participation will involve the completion of questionnaires, muscle endurance testing, and the possible participation in a 12-week rehabilitation programme which will consist of two exercise sessions a week and an educational session once or twice a week. You will be allocated into one of two groups for participation over the 12 week time period. This allocation is random and you are asked to accept your allocation position.

The questionnaires that you will be given to complete will be used to measure the following perceptions:

- the intensity of your own back pain (Visual analog scale)
- how your back pain impacts your activities of daily living (Oswestry disability index)
- the extent to which your back pain influences your activities of daily living (Functional rating index)
- how you perceive that activities like bending would affect your back pain (Fear avoidance beliefs questionnaire) and
- how afraid you are that activities and exercise might further injure your back (Tampa scale of Kinesiophobia).

The physical tests that will be used will consist of the following:

- a straight leg raise test (subject lies on their back and examiner lifts one leg and assess the amount of stiffness in the hamstrings and lower back)

- the Sorenson back extensor endurance test (examiner holds down your legs while the upper body hangs off the edge of an examining table in the face-down position, the upper body has to be kept parallel to the floor without support for the maximum amount of time)
- lower back active range of motion tests (subject is in the standing position, will be asked to bend forward, backwards, to the sides and to twist around, and the examiner will assess the amount of discomfort in the lower back)
- the Slump test (subjects sits off the edge of a table, hands are placed behind the back, chin is placed on the chest and the legs are extended one at a time by the examiner to assess the amount of discomfort in the hamstrings and lower back)
- the side-bridging test (subjects supports themselves on the ground on one forearm and on the feet while in the side lying position, the body is then lifted up and the straightened positions has to be held for as long as possible, and it will be done on both sides) and
- the 60° flexor endurance test (subject will sit on an examining table with the knees bent and the back supported so it forms a 60° angle, after which the support will be removed and the position has to be kept for as long as possible).

All of these tests have been selected because they place the least amount of pressure on the back, and are thus comfortable to perform, even though you suffer from low back pain.

After the tests have been completed, you will be allocated into either the conservative or progressive exercise groups.

- The conservative group will be asked to perform exercises that are considered conservative, non-treating and non-exertional by the academic experts. The exercises will be performed two times per week at the Research laboratory at LC de Villiers Sport Centre at the University of Pretoria and it will stay the same for the full 12 weeks of the study. Along with this, if you are allocated to this group you will also receive an information pamphlet that will give information on back safety and risky activities that should be avoided.
- The progressive exercise group will perform exercises that are more difficult than the conservative group exercises, but are still not very exertional in nature. After every 4 weeks, the exercises will be progressed to the next level of difficulty. This will be done for the full 12 weeks of the study. The exercises will be performed 2 times per week at the Research laboratory at LC de Villiers Sport Centre at the University of Pretoria. Along with this, this group will also receive “back school”, where for 15 minutes, there will be given an information lecture on aspects of low back pain, for example how to properly bend down to pick objects up from the floor. This group will also receive the information pamphlet that the other group receives. The exercise session and the “back school” session will be an hour in length. The questionnaires and the physical tests will be performed at certain intervals to assess the progress of both groups as they move through the rehabilitation process.

After the 12 weeks, both groups will receive a standard maintenance exercises programme to be performed at home that will be aimed at maintaining the effect of the exercise programmes. This will last for 6 weeks, after which both groups will again be issued with the questionnaires and physical tests to see if the effect has been maintained.

### **RISK AND DISCOMFORT INVOLVED**

There might be some slight discomfort involved during the exercise testing and the possibility of slight muscular stiffness afterwards, but this will be minimal and you are asked to willingly accept this possibility. The possibility of injury will be limited as best possible. Medical personnel will be at close proximity to all venues used, and they will be available at all times. However, there is a large risk involved if you did not seek medical advice about your back in the past, and the back pain is without a diagnosis. This study is only applicable to those suffering from non-specific low back pain, and all other causes have been eliminated. There is a very definite risk of paralysis if something is unknown and you proceed with the exercise programmes. Medical advice is a prerequisite for participation in this study.

### **POSSIBLE BENEFITS OF THE STUDY**

Remedial exercise therapy has been successful in the treatment of chronic low back pain. You will be receiving proven treatment methods if you have low back pain. **This type of treatment can become expensive, but you are receiving all of the treatments for free.** The conservative exercise programme has been shown to be successful in the treatment of chronic non-specific low back pain, and is thus a valid tool in the treatment of this type of low back pain. We hope that the progressive exercise programme can be even more successful and effective, and we hope that we can show this with this research project. Both programmes thus have potential as successful treatment methods, so being allocated to either group has no disadvantage. Thus, being in the conservative group is not a disadvantage in anyway, as this type of exercise programme is a recognized, valid and successful means of treatment.

### **I FULLY UNDERSTAND THAT I CAN WITHDRAW FROM THE STUDY WITH NO REPRIMANDS OR PUNISHMENTS.**

### **HAS THE TRIAL RECEIVED ETHICAL APPROVAL?**

This clinical trial Protocol was submitted to the Faculty of Humanities and the Faculty of Health Sciences Research Ethics Committee, University of Pretoria and written approval has been granted by those committees. The study has been structured in accordance with the Declaration of Helsinki (last update: October 2000), which deals with the recommendations guiding researchers in biomedical research involving human subjects. A copy of the Declaration may be obtained from the investigator should you wish to review it.

### **INFORMATION**

If you have any questions concerning this study, you should contact:  
Johnny Billson (082 612 0790) - researcher.

### **CONFIDENTIALITY**

All records obtained whilst in this study will be regarded as confidential. Results will be published or presented in such a fashion that patients remain unidentifiable.

### **CONSENT TO PARTICIPATE IN THIS STUDY.**

I have read or had read to me in a language that I understand the above information before signing this consent form. The content and meaning of this information have been explained to me. I have been given opportunity to ask questions and am satisfied that they have been answered satisfactorily. I understand that if I do not participate it will not alter my management in any way. I hereby volunteer to take part in this study.

I declare hereby that I will not withhold any information that could exclude me from participating in the research project, and I am aware that I am entitled to withdraw from the project at any time if I should wish.

I hereby also grant the researcher permission to use my results for publication and/ or presentation purposes, with my anonymity being ensured.

---

Signature of prospective participant

---

Signature of researcher



Tel: \_\_\_\_\_ (h) \_\_\_\_\_ (w)

Witness

1. \_\_\_\_\_

**EXERCISE PROGRAMMES:**



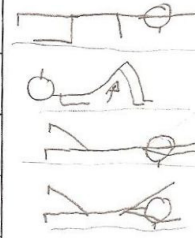
• **Control group exercises:**

Exercise	Sets	Reps
Cycling	5min	
Single leg to chest stretch	2	12sec
Both legs to chest stretch	2	
Hamstring stretch (hold back of knee, bend knee slightly)	2	12sec
Figure 4 stretch	2	12sec
Roll both knees to one side stretch	2	12sec
Pelvic tilting	3	10
Sit on stability ball	3	12sec





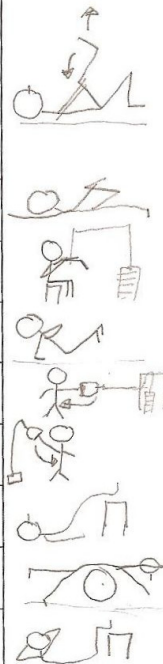
Alt superman on all fours	2	4
Hip lifts (feet flat on floor)	2	6
Prone alt leg lifts	2	6
Prone alt arm & leg lifts	2	6
Walking	10min	



• **Experimental group exercises:**

**1<sup>st</sup> Programme:**

Exercise	Sets	Reps
Cycling	5min	
Hamstring stretch (hold leg with both hands, bend leg slightly, perform 20 plantar/dorsiflexion step-offs with extended leg)	3	20
Side lying quadriceps stretch	3	12sec
Lat pulldown to front	3	15
Side bridging (on knees)	3	30sec
Cable Horizontal adduction	3	15
High cable horizontal adduction (downwards)	3	15
Hip lifts with feet on bench	3	15
Alt superman prone on stability ball	3	12
Abdominal crunches (feet on bench)	3	20
Cycling	5min	

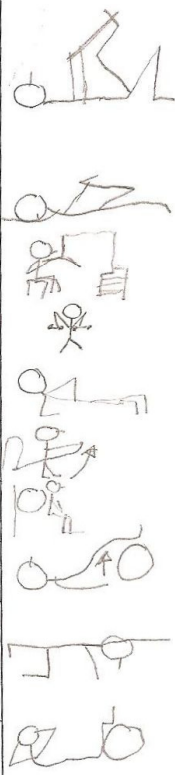






**2<sup>nd</sup> Programme:**

Exercise	Sets	Reps
Cycling (2 levels higher)	5min	
Hamstring stretch (hold leg up with towel, stretch for 12sec, 12 plantar/dorsiflexion step offs with leg in extended position, 12sec hold)	3	12;12;12
Side lying quadriceps stretch	3	12sec
Lat Pulldown to front	3	15
DB upright row	3	15
Side bridging (on feet)	3	20sec
Low cable shoulder extension (straight arm, knees bent)	3	15
Ball squat against wall	3	15
Hip lifts (feet on ball)	3	15
Alt Superman (sweeping hand on floor upon return and up again)	3	12
Abdominal crunches (feet on stability ball)	3	25
Cycling (2 levels higher)	5min	



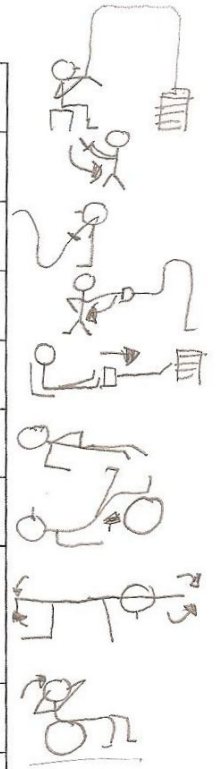
**3<sup>rd</sup> Programme:**

Exercise	Sets	Reps
Cycling (increase by 2 levels)	5min	
Hamstring stretch from programme 1	2	15sec
Hamstring stretch from programme 2	2	15sec
Side lying quadriceps stretch	2	15sec



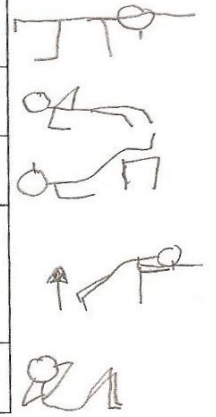


Lat pulldown to front	3	15
High cable pulldown to opposite hip with both arms	3	15
Cable upright row	3	15
Cable lateral raise from front	3	15
Horizontal leg press	3	15
Side bridging (On feet, roll on arms to other side)	3	20sec
Hip lifts with one leg at a time (feet on ball)	3	10
Alt superman (hold arm and leg at end range of motion and perform flexion/extension movements with hand and foot)	3	12
Abdominal crunches (lying on ball)	3	30
Cycling for (intensity has to higher than in 2 <sup>nd</sup> programme)	5min	



• **Maintenance Programme:**

Exercise	Sets	Reps
Alt superman sweep	3	12
Side bridging (On feet)	3	30sec
Hip lifts with feet on a chair	3	15
Back extension on kitchen table (lifting both legs at the same time while the upper body is lying on the table)	3	12
Abdominal crunches with feet on floor	3	30



## **Annexure D**

### **Case Study Reports**

Subject A

Subject B



### Visual Analog Scale (VAS)

Please indicate the amount of pain recently experienced by marking an (X) through the line.

100mm VAS scale – Left hand marker “no pain”, right hand marker “extreme pain”.



I have experienced this programme to be extremely effective. When I first started, I could even sit or stand without some form of pain, however after the 1<sup>st</sup> exercise programme I already felt an improvement. After the second programme I had little to no pain. This experience has improved my quality of life, and given me the tools to keep looking after my back. The back school part was extremely informative!

Thank you very much!



### Visual Analog Scale (VAS)

Please indicate the amount of pain recently experienced by marking an (X) through the line.

100mm VAS scale – Left hand marker “no pain”, right hand marker “extreme pain”.



→ Ek het geker om my postuur te verbeter my pelvis in te druk & my maagspiere sterk te maak.

→ Dit te bestuur is vermy in ongelooflike verbetering en ek hoef nie meer anti-inflammatoriese middels te gebruik op laas ritte.

→ my gemak-level is baie hoër en ek voel baie beter oor my rug.